



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

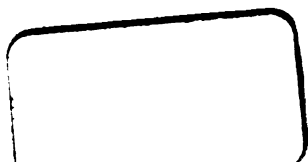
About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

No.

BOSTON
MEDICAL LIBRARY
ASSOCIATION,

19 BOYLSTON PLACE.



THE
CLINICAL JOURNAL.

*A WEEKLY RECORD OF CLINICAL MEDICINE AND
SURGERY, WITH THEIR SPECIAL BRANCHES.*

IN TWO VOLUMES ANNUALLY.

VOL. VII.

NOVEMBER, 1895—APRIL, 1896.

FOURTH YEAR.

EDITED BY

M. I. DENTON-CARDEW,

AND

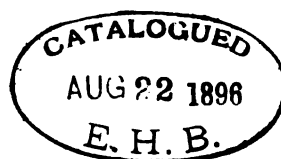
L. ELIOT CREASY, M.R.C.S., L.R.C.P. Eng.

OFFICES:

30 & 31, TEMPLE CHAMBERS, LONDON, E.C.

MDCCCXCVI.

The Clinical Journal, April 22, 1896.



LIST OF CONTRIBUTORS.

	PAGE
ALLCHIN, W. H., M.D., F.R.C.P. The Nature of Disease	70
BALDWIN, G. R., F.R.C.S. Tubercular Arthritis	209
BALL, J. B., M.D., M.R.C.P. Throat Cases	365
BARKER, A. E., F.R.C.S. Perforating Gastric Ulcer	361
BARLING, G., F.R.C.S. Perforation of Stomach	79
BARLOW, T., M.D., F.R.C.P. Herpes	311
BARWELL, R., F.R.C.S. Hip-joint Disease	347
BLACKER, G. F., M.D., M.R.C.S. Eclampsia	10
BOXALL, R., M.D., M.R.C.P. General Management of Childbed and Labour	165, 185
BRADFORD, J. R., M.D., F.R.S. Disseminated Sclerosis	323
BRAINE, C. C., F.R.C.S. Fatal Case of Hæmorrhage under Anæsthesia	359
BRODIE, C. G., F.R.C.S. Congenital dislocation of the Hip	84
BRYANT, T., F.R.C.S. Clinical Cases	112
BUTLIN, T. H., F.R.C.S. Treatment of Cancer by Injection	213
BUXTON, DUDLEY, M.D., M.R.C.P. Pental as Anæsthetic	342
CHEADLE, W. B., M.D., F.R.C.P. Clinical Notes	386
CLARKE, J. M., M.D., M.R.C.P. On Apoplexy	135
CLARKE, J. JACKSON, F.R.C.S. Cocaine Anæsthesia	175
Mastoid Abscess	278
COUPLAND, S., M.D., F.R.C.P. On Typhoid Fever	45, 61, 77
CRAIG, M., M.B., M.R.C.S. Alcoholic Insanity	329
DORAN, A., F.R.C.S. After Treatment of Abdominal Section	251
DUFFIN, A. B., M.D. On Acute Primary Myelitis	1
ECCLES, W. MCA., F.R.C.S. Anomalies of Testes in Relation to Hernia	286
GALABIN, A. L., M.D., F.R.C.P. Hysterectomy	102
GOWERS, W. R., M.D., F.R.S. Anterior Poliomyelitis	241
Caries of Spine	17, 33
Poliomyelitis, Adult	313
GRANT DUNDAS, F.R.C.S. Diagnosis of Pain in and about the Ear	5
GUNN, R. M., F.R.C.S. External Examination of the Eye	137
HABERSHON, S. H., M.D., F.R.C.P. Clinical Cases	397
HALL, A. J., M.R.C.P. Exophthalmic Goitre	38
HALL, F. DE H., M.D., F.R.C.P. Gastric Ulcer	161
HARLEY, GEO., M.D., F.R.S. Formation and Structure of Calculi	30
HEATH, C., F.R.C.S. Common Diseases of Rectum	65
Stricture of Rectum	202
HERMAN, G. ERNEST, M.B. Lond., F.R.C.P. On Diseases known as Endometritis ..	377
HORSLEY, VICTOR, F.R.C.S. Traumatic Neurasthenia	281
HUTCHINSON, J., LL.D., F.R.C.S. Demonstrations at Clinical Museum	28, 254, 321, 357
JAMES, A., M.R.C.S. Pontine Lesions	125

	PAGE
JAMISON, W. A., M.D., F.R.C.P. Psoriasis	154
LACK, H. L., M.D., F.R.C.S. After Treatment of Tracheotomy	227
LOCKWOOD, C. B., F.R.C.S. General Septic Peritonitis	349
MACCORMAC, SIR W., M.A., D.Sc., F.R.C.S. Diseases of Testicle	149, 177
New Growths of Penis	130
MARTIN, S., M.D., F.R.S., F.R.C.P. Early Phthisis	302
Treatment of Diphtheria by Antitoxic Serum	383
MILLER, A. G., M.D., F.R.C.S. Organic Stricture of Urethra	21
MORGAN, J. H., F.R.C.S. Lithotripsy in Two Cases of Recurrent Calculus	369
PAGET, S., F.R.C.S. Surgical Cases... ..	35
PHILLIPS, J., F.R.C.P. Clinical Cases	75
POORE, G. V., M.D., F.R.C.P. On Chronic Lead Poisoning	193
Clinical Notes	393
QUAIN, SIR R., F.R.S. Reminiscences	51
ROBERTS, F. T., F.R.C.P. Heart Disease	27
ROBSON, A. W. M., F.R.C.S. Sarcoma of Kidney	98
ROLLESTON, H. D., F.R.C.P. Theory of Jaundice	122
ROUTH, A., M.D., M.R.C.P. Uterine Hæmorrhage	117
RUSSELL, J. R., M.D., M.R.C.P. General Paralysis of the Insane	355
SEMON, F., M.D., F.R.C.P. Malignant Disease of Larynx	265
SMITH, R. S., M.D., F.R.C.P. Morbus Cordis with Hepatic Enlargement	145
SNELL, S., F.R.C.S. Use of Mydriatics in Eye Examination	41
SPENCER, H. R., M.D. Ruptured Perineum	307
SUTTON, J. B., F.R.C.S. Central Tumour of Spinal Cord	14
TUKE, T. S., M.B. Lunacy and General Practice	171
WALDO, H., M.D. On Chorea	15
On Myelitis	109
On Ascites	289
WALSH, Surg.-Capt. Dysentery	261
WARNER, F., M.D., F.R.C.P. Ulcerative Endocarditis	257
WATSON-WILLIAMS, P., M.D. On a Case of Graves' Disease	93
WEST S., M.D., F.R.C.P. Alcoholic Delirium	58
WHITE, W. HALE, M.D., F.R.C.P. Thoracic Aneurysm	297
Peritonitis	317
WILKIN, G. C., M.R.C.S. Aural Polypi	339

THE CLINICAL JOURNAL.

3906

WEDNESDAY, OCTOBER 31, 1895.

AUG 32 1896

A CLINICAL LECTURE

ON

ACUTE PRIMARY MYELITIS.

Delivered at King's College Hospital on May 11th, 1895,

By A. B. DUFFIN, M.D.,

Physician to the Hospital.

I DESIRE to-day to lay before you the histories of two cases of primary acute myelitis of the spinal cord. I say primary because we are able with considerable confidence to exclude all ordinary sources of extrinsic pressure such as disease of the vertebræ or tumour. The first case is one of a very limited transverse lesion in the mid-dorsal region. The second, which you have had the opportunity of watching, is one of very extensive myelitis, rapidly spreading so as to involve the cord almost up to the origins of the phrenics.

A gentleman of some 35 years of age had to go over during the winter months to Paris on business. On the steamer he got into a berth, the small window of which happened to be open. He lay for some five or six hours with his back close to this. When he left the ship his back felt stiff and numb, but there was nothing unusual to attract his attention. When he reached Paris and walked to a cab his feet felt numb and tingling, and the mid-dorsal region of his back ached decidedly. He, however, got into his cab, and after depositing his luggage at his hotel, drove to the Place de la Concorde. There he dismissed his vehicle and tried to walk. He at once noticed that his legs dragged, and having got with some trouble into the main roadway, he had the greatest difficulty in lifting his foot on the curb. He became alarmed, and at once drove back to the hotel. There he had to be assisted out of the vehicle and carried to his bedroom. He determined to return to London by the next train. As soon as he reached his own home he telegraphed to me.

I found him completely paraplegic as regards motion in both legs. His sensibility was impaired,

Vol. VII. No. 1.

but present. Both the superficial and deep reflexes were markedly exaggerated. He could empty his bladder, but with a feeble current. He had distinct girdle pain, with a hyperæsthetic zone of no great width or intensity running through the umbilical region, with loss of the umbilical reflex. The "rheumatic" pains in the back had almost gone, and he could bear the vertebræ to be freely handled without distress. Rotation of the spine was also well borne. A temperature rise of very moderate amount.

I wet-cupped his mid-dorsal region, and subsequently applied moist warmth. I kept him strictly at rest, and gave him full doses of iodide of potassium.

The most satisfactory results followed. First his subjective sensations in the limbs ceased, and normal ones returned; then his girdle discomfort left him. His power over his bladder next returned; and lastly, all his loss of motility was regained. All these results ensued in about ten days.

The man, unfortunately, believed himself cured. He had the folly to walk from his home, a distance of over two miles, to his City office. He was brought home that day paraplegic, and he has remained so ever since. His second attack was unattended by any modification of sensibility in the lower limb whatever, and his girdle pain required the use of a hot sponge to detect it. About a year subsequently secondary rigidity set in and became very marked. As his legs had been kept in good extension and with the feet at a good angle, the secondary rigidity fixed the limbs in a favourable attitude, and with a couple of sticks he was able to move about to a really remarkable extent.

Here, then, we have a case of primary acute myelitis due to direct exposure to cold. This inference we found upon the rapid appearance, first of subjective sensations, and then of rapidly deepening loss of power supervening on the exposure. We say transverse because the condition of the reflexes gives us direct evidence that the segment of the cord below the lesion was healthy. The reflexes, both superficial and deep, were

markedly exaggerated. In which part of the cord vertically was the lesion situated? This, the zone of modified sensibility, passing through the umbilicus, enabled us to determine, even irrespectively of the fact that we know the mid-dorsal region to have been exposed to the open window, and to have been for some twenty-four hours subsequently the seat of rheumatic pains. As regards the extent of the cord involved, I do not think it was vertically of any great dimensions; the girdle zone was a narrow one, and the umbilical reflex was the only one lost. As regards the transverse amount of the damage, it was chiefly on the antero-lateral motor columns that the mischief fell. Sensation was only slightly and for a short time impaired. That the multipolar cells escaped we infer from the fact that no wasting followed. That the initial lesion throughout was a slight one is evident from the rapid and complete recovery which at first ensued. The secondary rigidity, which subsequently proved the patient's best friend, we interpret to mean secondary descending sclerosis invading the antero-lateral columns from the site of the lesion.

The history in this particular example is so convincing of a primary inflammatory origin that no question of any locally compressing cause can arise. But by far the majority of cases of localized transverse myelitis are due to slow compression either from tumour or vertebral disease.

L. B., 17, Housemaid.

Admitted April 29th, 1895.

Complains of numbness and complete loss of power in the lower extremities and body as high as the mammæ; also of severe pain in the right breast, pain of a tingling character, and partial loss of power in both arms and hands, headache, inability to retain either urine or fæces.

The family history and personal antecedents present nothing remarkable.

Early in January last, in consequence of bursting of water-pipes, patient was exposed to a severe chill. In the earliest days of March last she for the first time noticed tingling pains in the right foot and leg to the knee, alternating with a burning sensation over the same area.

Patient was, nevertheless, able to perform her duties up to April 20th, i.e. nine days before

admission. She then found that she could not wear her stays owing to a severe pain in her right side, just below the ribs. At the same time she became absolutely constipated. On April 21st, when attempting to rise from bed, she noticed that both feet and legs were quite numb, and that she could only stand upright with assistance. She felt generally ill. The next day control over the bladder was lost, and the catheter was required. By the 24th the loss of sensation had become absolute as high as the lower ribs, and motor power had quite vanished. Both mammæ had also become very tender, and the shoulder movements were painful. On the 27th she found the movements of the right arm and hand to be somewhat restricted, and the left arm also, after a few hours, became enfeebled. Both arms and hands became numb and tingling. 29th, admitted.

Physical Examination. In front, all forms of sensation are lost from the toes to the lower edges of the mammæ, terminating in a sharply-defined line corresponding to the sixth intercostal space on both sides. From this line a zone of hyperæsthesia extends almost to the root of the neck, but is most intense on the right side and between the third and sixth ribs. There is also very slight hyperæsthesia of both arms.

On the back anæsthesia extends as high up as the angles of the scapulæ, ending in two sharply defined lines which run up to a point opposite the third dorsal spine. Hyperæsthesia extends from this point to the vertebra prominens, or a trifle beyond it. *Motility.* Facial movements, as also those of rotation and nutation of the head are unaffected. *Left arm.* Feeble adduction, abduction, flexion, and extension of shoulder. Elbow permits of flexion, but extension is very imperfect; wrist feeble supination, pronation; flexion and extension somewhat limited. Movement of metacarpo-phalangeal joints limited; phalanges quite lax, are kept in semiflexion. *Right arm.* The movements of the shoulder-joint are all very poorly represented, but present; those of the elbow are also feebler and more restricted than those of the opposite left joint. All the movements below the wrist are extremely circumscribed and feeble, while those of the fingers are quite lost. She can shrug both shoulders fairly well, but the movement is best marked on the right side. *Lower extremities.* Absolutely no movement of

any sort can be obtained, the feet are "dropped," and all the muscles are completely relaxed.

The abdominal muscles and the intercostals up to the third space are quite powerless. Some scanty costo-superior breathing exists, and the diaphragm continues to act. The muscles of the legs do not react to faradism, but there is hitherto no appreciable wasting.

All the superficial and deep reflexes as high as the mid-thorax, and including the scapular, are completely lost.

The bladder is totally paralyzed, and there is overflow incontinence. The rectum is also paralyzed. The urine is ammoniacal; there is free, bright hæmorrhage from the bladder, and an extensive deposit of pus and phosphates. The catheter is now freely used.

There is an extensive sloughing bed-sore, with angry edges over the sacrum and lower lumbar region.

The heart sounds are normal. Breathing is shallow, almost absent over the lower thorax, but at present vesicular and free from mucous rales. Liver and spleen of normal size. Temperature averages 103° , but oscillates between 104 and $99\frac{1}{2}$. Face is flushed, but aëration is good.

Patient was ordered to take thirty drops of oil of turpentine in capsules as a hæmostatic daily. Her bladder was twice daily washed out with 1—30 sanitas, and subsequently injected with equal parts of hazeline and water, a.a. three ounces. The bed-sore was dressed with charcoal poultice, smeared over with ungt. resinæ. Patient was placed on a water bed. Three days later the bed-sore had cleaned, and was looking healthy, the hæmorrhage from the bladder had quite ceased, although the urine still contained a heavy deposit of pus and phosphates. On *May 5th*, i.e. seven days after admission, the hyperæsthetic zone had left the mammae, had risen two intercostal spaces, and was now most severe about the right shoulder joint. It had also extended up the back of the neck as high as the fourth cervical spine. For the first time she became delirious at night. On *May 10th* the bed-sore was found to be again extending, with fresh sloughs and very angry edges. Iodoform was freely applied. In the urine the pus and phosphates had materially decreased, and the secretion was acid. The area of hyperæsthesia had not materially altered. During last

night she has had three attacks of most ominous dyspnoea, but it was noticed that her diaphragm continued to act well while they lasted.

In what way does this case differ from the first? If we can accept the subjective sensations first experienced in early March on the starting point of the disease, the mischief seems to have begun in the lowest segment of the cord and to have remained for some six weeks localized to a very small centre. But from April 20th evidence of rapid extension supervened. In the course of a few days the whole cord up to the upper thoracic region became involved. In this case we had no transverse damage with sound cord below it. This we infer, firstly, from the complete absence of reflexes from the thorax downwards, the absolute loss of sensibility, the marked foot drop and permanent flaccidity of the muscles with absence of faradic reaction. This condition of muscle is strong evidence that the lumbar intumescence has been gravely affected. In addition we have acute and spreading trophic bed-sore, the complete paralysis of bladder and rectum, and the evidences of acute inflammation of the bladder. In the first case all these things were absent, and I especially noted that the reflexes, both superficial and deep, were markedly exaggerated. The mischief in this poor girl, we have reason to believe, is rapidly creeping upward. It has already involved all the respiratory muscles except the diaphragm and the upper intercostals. The sensitive branches of some of the higher cervical nerves and some of the spinal roots of the spinal accessory are probably already implicated. Any day the centre for the phrenics may become involved and life rapidly extinguished by apnoea. The cervical intumescence is thus already extensively involved. In how far may the very high and oscillating temperatures we have noted have any relations to this fact? It is well known that surgical injuries to the cord very high up have been followed by paradoxical temperatures either hyperpyrexial or collapse. But here we have the extensive bed-sores and the septic absorption to which they give rise, to explain the character of the fever.

The course in this example has been, and will probably continue to be, a rapid one, the first decided advance only dating back one month. It is swift even for the class of extensive spreading myelitis to which it belongs.

In simple transverse myelitis, and especially in those cases where the motor tracts are exclusively, or, at any rate, mainly attacked, the case is different. If taken very early even complete recovery is possible. Partial recovery, first of sensation and then of more or less motor power is not uncommon, but most usually at the end of a few weeks or months secondary sclerosis attacks the antero-lateral columns at the site of lesion and descends through the motor tract of the cord. Improvement is then replaced by secondary rigidity and the joints become more or less fixed. If this has been thought of during the time the muscles were lax, it is far from an unmixed evil, as the patient may be able to work about fairly well on the stiffened limbs. But the stiffening once acquired is permanent. Sometimes a further accident breaks in.

The anterior-multipolar cells also become slowly involved in the sclerosis, and secondary wasting of the affected muscles results. The first indication we usually get of this is that the deep reflexes, which have been hitherto greatly exaggerated, fail to respond. Then the muscles become again rather flabby to the hand, faradic excitability is lost, and the reaction of degeneration develops to voltaism. The poor patient may thus again lose his power of walking, and become for the second time fixed to his couch. A third accident that may complicate acute transverse myelitis is the extension of the mischief right down the cord. In such cases the deep reflexes are not only completely lost, but sensibility is also forfeited, fever becomes severe, and acute bed-sore and bladder troubles supervene. We get, in fact, by two stages, with a more or less marked interval, into much the same state as is presented by the girl up-stairs.

The subsequent course of this second case runs as follows.

Two days later (May 12) there was marked hyperæsthesia of the fingers, and motility in the upper extremities seemed quite limited to the shoulder joint, and very painful there. Acute œdema of the left leg has set in, although no thrombosed vein could be found. Her subsequent story was that her breathing became more distressing, she became dusky, and her chest rapidly filled with mucous rales. Patches of gangrene showed themselves on the malleoli and heels.

The urine, remarkably enough, kept acid to the end, and the last specimen obtained only had a trace of albumen. She died during the night of the 16th.

For the notes of the post-mortem examination, I am indebted to my colleague, Prof. Dalton.

The lungs, besides being œdematous, contained scattered patches of pneumonia. Both kidneys showed purulent pyelitis, with small abscesses in the kidney substance. Bladder acutely inflamed. The sacral bed-sore had extended into the pelvis, so that a gangrenous cavity lay to the right of the uterus, but had not perforated the peritoneum.

CORD.—The *upper part* of the cervical intumescence showed sclerosis of the posterior-median column. Under the microscope, this was found most marked in the anterior portion of the column. The central canal was filled with leucocytes and débris, but the epithelial lining was perfect. The multipolar cells in the anterior cornua of this area, with a few exceptions, retained their shapes, but their nuclei were extremely degenerated, although the nuclei of the connective tissue around were perfectly normal.

In the *middle* of the cervical enlargement acute softening occupied the anterior half of the postero-median and postero-lateral columns, and the area round the central canal of the cord. It was quite symmetrical. In this area all the cornua were still distinct to the naked eye. Microscopically, all the parts at this level were necrotic, and a general infiltration with leucocytes could be made out. In the *lower* part of the cervical enlargement the soft area was quite diffuent, and extended more to the left side, destroying the left posterior cornu. Just below the cervical enlargement the softening was so great that only the right anterior cornu could be at all recognized. Below this again the softening became so extensive that no parts could be differentiated down to the very end of the cord.

Coryza. (*Bull. Pharmacy*):

R	Salol.	1 part
	Acid Salicyl.	20 parts
	Acid Tannic	10 parts
	Acid Borac. (Pulv.)	4 parts

M. One pinch should be taken into each nostril at the commencement, and then every hour for eight hours, *but not longer*.

LECTURES
ON
**THE METHODICAL DIAGNOSIS OF
DISEASES CAUSING PAIN IN AND
ABOUT THE EAR.**

Delivered at the Central London Throat and Ear Hospital,
Gray's Inn Road, February 26th, 1895.

By DUNDAS GRANT, M.A., M.D., F.R.C.S.,

Surgeon to the Hospital.

(Reprinted from shorthand notes.)

GENTLEMEN,—I need hardly say that pain, as a symptom of ear-disease, is one of the most serious we meet with; while its importance to the patient is, of course, beyond question. The first thing he desires is to be relieved of his pain, but it is also important to us, from a diagnostic point of view, on account of the serious conditions of which it is only too often the index.

For our present purpose it will be useful to divide pains in and around the ear into three groups:—

- A. Those centred in the ear.
- B. Those centred in the mastoid process.
- C. Those which are more generalized over the head, and accompanied by febrile or cerebral disturbance.

A. Now, certain of those which are SITUATED IN THE EAR are—I. Unaccompanied by any form of inflammatory disease in that part, namely the neuralgias (or otalgias, as we term them). We diagnose these when we find there is neither deafness nor any local sign of inflammation. The pain may be either reflex, or not; it may be either neuralgia of the fifth nerve of the cervical plexus, or of the tympanic plexus. You doubtless know the details of the innervation of the external ear. The external auricle is supplied by the auriculo-temporal nerve; it is also supplied by the great auricular nerve from the superficial cervical plexus. A large portion of the outer surface of the pinna is supplied by the auriculo-temporal nerve, as is also the greater part of the meatus, but certain portions of the pinna, namely, the antitragus and part of the helix, are supplied by the great auricular from behind. The posterior portions also are served by the great auricular. The back of the

concha is supplied by Arnold's nerve, a branch of the vagus, which comes out between the meatus and the mastoid, and supplies portions of the inner surface of the ear, also a portion of the concha. There is a curious part of the distribution which is often forgotten, namely, that the upper part of the auricle is supplied by the small occipital nerve, a branch of the superficial cervical plexus. The meatus is served chiefly by the auriculo-temporal, but also by Arnold's nerve, a branch of the vagus, which is answerable for the occurrence of cough when the interior of the meatus is irritated, whether by a foreign body or by the presence of cerumen. The middle ear itself is chiefly supplied by the tympanic plexus, but the auriculo-temporal nerve supplies the membrana tympani. The tympanic plexus, you will recollect, is formed by Jacobson's nerve from the glosso-pharyngeal by a communicating branch from the carotid plexus and by the small superficial petrosal. This latter, you will remember, communicates with the third division of the fifth through the otic ganglion. There is a very intimate connection in the ear between the glosso-pharyngeal and fifth nerves.

We find that otalgia of the fifth nerve may be *reflex*, and occasioned, as it most often is, by disease of the teeth; it may also arise from disease of the throat, for in cancer and other affections of the larynx or tonsils the pain frequently shoots up into the ear. That would be reflex otalgia of the fifth through the auriculo-temporal. Then, occasionally, we have a reflex otalgia from new growths affecting the cervical plexus. But supposing that no reflex cause is discoverable, and the pain is situated deeply in the ear, it is *tympanic neuralgia*, intrinsic to the tympanic plexus itself, which, when there is no cause apparent to account for reflex otalgia, you may be often at a loss to diagnose. You have then to think of such cause as malaria and syphilis (for which you must have further corroboration, and that especially when the pain is greater at night). Again, there may be gout, or toxic affections of one kind or another; or there may be malignant disease inside the skull or in the region supplied by the fifth nerve. You are further assisted in your diagnosis by being able to localize the tender points. For instance, in the superficial cervical plexus we find the tender points down the back of the sterno-mastoid and in the parietal region. Again, in neuralgia of the fifth

nerve, you may find the tender points, if it is in the third division, where the inferior dental nerve emerges. Or, in the case of the second or of the first division, pain at the side of the nose, or in the supra-orbital region. In general, we may say that in the absence of deafness or signs of inflammation in the ear, pain in that situation may be generalized as otalgia.

II. Supposing there is a MODERATE DEGREE OF DEAFNESS; that the deafness is NOT INITIAL; that is to say, it has not preceded or accompanied the pain from the beginning; then we may safely say that the pain does not arise in the tympanum itself, because any inflammatory condition in the tympanum giving rise to pain would be associated with very great diminution of hearing. We have to deal with one of the forms of *acute external otitis*. If the pain is comparatively moderate, and the swelling uniform, the disease is *acute diffuse external otitis*. I should add that it is occasionally hæmorrhagic, that is to say, people are occasionally attacked with pain and the symptoms I have mentioned, and in addition, have a narrowing (as shown here) attended by the formation of blebs containing blood; these burst and cause the outpouring of blood, and the disease is then known as *external hæmorrhagic otitis*.

But suppose the pain is intense, and paroxysmal, so as to make certain hours of the night perfectly miserable (remember the deafness is not initial), and that the swelling is localized to one of the walls of the meatus, then the affection is *acute circumscribed external otitis*, otherwise "furuncle of the ear." This is often superposed upon eczema, or upon chronic diffuse otitis, the furuncle being excited by slight traumatism, as for instance by the finger-nail, on account of itching, or by the unskilful insertion of instruments into the ear.

Now suppose the congestion, instead of being diffused over the walls of the meatus, is confined to the outer surface of the membrana tympani, we have to deal with *acute myringitis*, and I show you here such an appearance as you may have in a comparatively slight attack. The congested membrane afterwards becomes swollen, and is then covered with detached epithelium, and when that is thrown off you may notice granulations upon the surface of the membrane from which the epidermic layer has been removed. Sometimes there is the formation of vesicles, even little

sacks formed by the outer layer of the membrane, and the drawing here is taken from Politzer's text-book. The same drawing may stand for acute, non-suppurative inflammation of the middle ear, and the only means of distinguishing one from the other is by the fact that the deafness does not come on at the commencement of the pain in simple myringitis; in fact, defect of hearing never attains any considerable extent, whereas in middle ear inflammation the lowering of the hearing is enormous, even though the appearance of the membrane betokens no further disease.

III. I now come to those affections in which THE DEAFNESS IS VERY CONSIDERABLE; that is to say, a watch would only be heard in contact, or within about an inch of the ear. Considerable deafness, which dates FROM THE COMMENCEMENT of the affection indicates *median otitis* in one of its acute forms.

If the pyrexia is comparatively slight and you find such an appearance as shown here, then you are dealing with *acute catarrh of the middle ear*, or it may be called *acute non-suppurative inflammation* of the middle ear, to distinguish it from the slight catarrh of the tympanum which accompanies catarrh of the Eustachian tube. The tendency of this is to get well, but sometimes it gets converted into that form in which the pyrexia is very great, and there is perforation at an early stage. As a rule it is a question as to the causes; possibly from the origin it may assume the severe form—*acute purulent inflammation of the middle ear*. This is peculiarly likely to occur when inflammation of the middle ear comes on in the exanthemata, such as scarlet fever, or measles, or when it accompanies diphtheria. I show you here the appearance ordinarily presented by a membrane when the perforation has just taken place. You see the perforation is somewhat black in appearance, and surrounded by a halo of redness, and the outlines of the membrana tympani have been lost. Before perforation occurs the membrane often presents the appearance illustrated here—entirely red to commence with, but as it begins to thin you may see what looks like a yellow pustule, showing that the pus is trying to find its way out.

Now, in acute inflammation of the middle ear sometimes a sack forms, such as I show you here, and that assumes a peculiar teat shape. Such cases are very troublesome, and are apt to lead to

affection of the mastoid cavities. Occasionally there is no question that suppuration of the middle ear is confined to the region of the attic, and the postero-superior quadrant of the tympanum. It is not often recognizable at the early stage, but we know, from the number of cases in which we find perforations in Shrapnell's membrane, with chronic suppuration, and a history of acute inflammation at some prior date that certainly suppuration may take place in the region. It is generally supposed that perforations take place invariably in the antero-inferior quadrant, and so it has always appeared to me, but Bezold has lately been asserting that such is not the case, and I am very strongly inclined to agree with him, because if it were the case we should not, so often as we do, find depressed cicatrices in the postero-superior quadrant, unless perforation very frequently took place in that region. Now the reason for it appearing to us that perforations take place in the antero-inferior quadrant, is that when the whole membrane is projected by means of pus in the interior of the tympanum (you will remember that the anterior part is very much further away from you than the posterior) swelling takes place, the posterior part bulges out, and completely hides, or nearly hides, the anterior and lower part, then if the perforation takes place on the lower part of the projecting portion of the posterior membrane, the pus appears to exude from down below. But if, when the inflammation begins to subside, you take a probe covered with cotton wool, and push the projection back, you will see that the perforation is really in the posterior half of the membrane, and we often find it there when things settle down.

Occasionally we find a form of acute perforative inflammation of the middle ear, which is of the *hæmorrhagic* form. I had accidentally left it out of my original plan for the lecture, but was reminded in a singular way. Of course it is very rare, and I confess I do not remember many cases, but it was brought to my attention only this very morning. On the Sunday before last, a gentleman awoke with a perception of deafness in his right ear. By one o'clock in the day he had a most frightful pain, and this was followed by a discharge of blood in considerable quantity. Next day the pain had completely subsided, but for the following two days he had still a little discharge of

blood, and the deafness persisted to some extent. Dr. Collier, of Oxford, sent him up to me on account of the deafness, and on examination I found the appearance I show you here; appearances of acute catarrh of the middle ear, but with a perforation covered by a little blood clot. His hearing is apparently improving very rapidly. That is hæmorrhagic median otitis.

In connection with pain limited to the ear, I ought to add that PAIN OCCURRING IN THE COURSE OF CHRONIC SUPPURATIVE INFLAMMATION OF THE MIDDLE EAR should always be a source of anxiety; it is very often the only symptom which will lead us to look for the evolution of one of the most severe processes. As a rule, the pain indicates either that a cholesteatoma is awakened into activity, or that some carious process is taking place; this caries may be affecting the tegmen tympani, and may lead, ultimately, to extra-dural abscess, and later to meningitis, or it may be a carious opening into the sigmoid groove, such as to allow of the setting up of sinus phlebitis. In any case, let me advise you to be anxious about cases in which attacks of pain come on in the course of chronic suppuration of the middle ear.

B. I now come to the second part of my subject—PAIN CENTRED IN THE MASTOID PROCESS. When pain affects this part, you may sometimes find a very evident cause for it; at other times you may not.

I. Let us first consider those cases in which there is EVIDENT MASTOID SWELLING, REDNESS, AND TENDERNES. This may present different aspects. In the first place there may be a circumscribed swelling over the mastoid, which you can move about with your finger. That you could easily realize as *inflammation of the mastoid lymphatic gland*. On the other hand there may be shelving swelling, which is due to *periostitis of the mastoid*, or (a more severe form) fluctuating swelling with projection of the auricle downwards, outwards, and forwards. That is a very important condition, and I show you here a rough sketch of a very extreme case figured by McEwen in his book. This *sub-periosteal abscess* is almost invariably connected with a focus of suppuration somewhere in the petrous bone; it may be in connection with the antrum, or with caries of the roof of the meatus; or it may even be in connection with abscess in the interior of the cranium,

which may find its way out through a little carious opening and form a sub-periosteal abscess over the mastoid. I daresay you recollect that in the development of the temporal bone the petromastoid and the squamous portions are joined together by a suture, which on the inner side is called the petro-squamous, through which, in children, disease may very often extend to the meninges of the brain. On the outside it is called the squamo-mastoid suture, which is generally completely closed at the end of the first year of life, though it sometimes persists later, and may allow communication of suppuration between the inside of the mastoid and the outside. Sometimes, indeed, there may be perforation in the squamous bone opening into the groove for the lateral sinus, and the extra-dural pus may find an outlet so as to form an abscess such as I have described.

II. In many cases of pain in the mastoid region we have no such marked swelling, and that gives me my second category, viz.: NO MARKED SWELLING, BUT DEEP-SEATED PAIN AND CONSTITUTIONAL DISTURBANCE. These indicate disease going on in the interior of the mastoid. When this comes on after acute otitis, the tendency almost invariably is for it to centre itself on one of the cells close under the surface of the mastoid, when you get *cortical mastoiditis*. If it follows chronic suppuration, it as certainly affects the *mastoid antrum*, and produces suppuration there. In treatment it is very important, because in the acute case we only open the superficial layer of the mastoid, whereas in chronic it is necessary to open the mastoid antrum itself.

III. We now come to the third category: INTERMITTENT PAINS OVER THE MASTOID PROCESS WITH NO LOCAL OR GENERAL SIGNS OF INFLAMMATION; that is to say, there is no constitutional disturbance, no tenderness on pressure, but pain coming and going, in short, *mastoid neuralgia*. That affection is certainly most frequent in those who have been subject to long-standing inflammation of the mastoid process, which has settled down. You can only discern it by the absence of local or general signs of inflammation.

C. I now come to the most serious form of malady with which the aural surgeon has to deal, and the class includes those fatal intracranial lesions which arise from acute or chronic suppurative inflammation in the middle ear. The PAIN IS MORE

OR LESS GENERALIZED OVER THE HEAD, WITH FEBRILE AND CEREBRAL DISTURBANCE, which may take the form of giddiness, or delirium, or vomiting. In the first place we ask ourselves whether it has been acute, or whether it has been chronic. Although there are exceptions to this wide generalization, there are certain forms of intracranial disease which follow the acute, and certain others which are consequent on chronic aural disease.

I. If it has been ACUTE, the first necessity is to ascertain whether there is any obstruction in the middle ear, and find out whether the cerebral disturbance and fever are relieved by evacuation of pus. If simple evacuation by washing out the meatus and opening the membrane or removing granulations and polypi is not sufficient, we have to resort to irrigation through the Eustachian tube, the details of which I have pointed out in another lecture. If that gives relief, we may say the symptoms have been due to the *retention of pus, etc.*, in the cavities of the middle ear. If, however, relief is not obtained by those means, then our first thought should be the possibility of mastoid implication. We should first try the effect of cold to the mastoid, and if speedy relief does not follow that the mastoid must be opened; this, as I explained, is a simple matter in an acute case. It often gives relief, and when it does so we know we have been dealing with *cortical mastoiditis*. But supposing even that operation does not give relief, we have to be prepared for two possible dangers, namely, 1, meningitis; 2, pyæmia. If there is CONTINUED PYREXIA (although the temperature may be going up and down it is still considerably above normal) and delirium, vomiting, constipation, photophobia, possibly squinting and drawing back of the neck, then we may be pretty certain that it is *meningitis* we are dealing with. Supposing, on the other hand, there is an OSCILLATING TEMPERATURE (going considerably above and below normal), and with rigors, generally on the rising side of the curve, and, more especially with metastatic suppuration in distant parts, then we are dealing with *pyæmia*. It is not common for people to die of acute aural inflammation, but it sometimes does happen, and when it occurs it is either from acute meningitis or acute pyæmia. The meningitis arises generally either from extension through the thin tegmen tympani to the membranes, or, it may be, and

probably generally is, caused by the passage of pathogenic organisms through the lymphatics. On the other hand, pyæmia usually takes place through the entrance of pathogenic organisms into the venous channels; but acute aural disease is not so likely to cause sinus phlebitis (phlebitis of the lateral sinus) as it is to affect the meninges straight away. However, it is a very rare occurrence.

II. Here, then, is a class of disease dangerous to life, resulting from CHRONIC aural disease, with suppuration. When we come in contact with such a case we have to think of it being simply due to retention of pus, or to disease of the antrum, or to extra-dural abscess, to meningitis, to pyæmia, sinus phlebitis, or to abscess of the cerebrum or cerebellum.

Suppose the temperature is continuously high, we think of retention of pus, antral disease, extra-dural abscess, or meningitis, but if the temperature oscillates tremendously, we think of pyæmia, sinus phlebitis. If we find the temperature subnormal after an initial rise, we think of abscess of the brain.

1. In the first place, let us suppose that the TEMPERATURE IS CONTINUOUSLY HIGH, possibly with most severe cerebral symptoms; the cause may be the retention of pus in the cavities of the middle ear. We have to try the effect of evacuation, and, if necessary, tubal irrigation. If relief follows that we know we have been dealing with simple *retention of pus*. If relief is not so obtained, if it is only obtained by opening the mastoid antrum, then we find it is due to *antral disease*, and very often to the formation of a *cholesteatoma* in the antrum, the natural history of which I described to you previously. I reminded you of its harmlessness if not aroused by damp or pathogenic organisms. When excited, cholesteatoma is one of the most serious ear-affections we have to deal with. If the opening of the antrum does not give relief, then, after a chronic case, we must think of the possibility of inflammation in the interior of the cranium, and in the first place of *extra-dural abscess*. This usually takes place above the tegmen tympani, so that if you were to trephine just above the osseous meatus you would find pus between the dura mater and the bone. Again by trephining a little further back than where you open the antrum you expose the lateral sinus, and it is not uncommon to find

pus there. If this treatment does not afford relief, the probability is that *meningitis* has to be encountered, when delirium, constipation, and the other symptoms I have mentioned are also present.

2. We will next suppose that the temperature, instead of being continuously far above normal, OSCILLATES from above to below normal, and is attended by repeated rigors, then, especially if you notice metastasis, you have to deal with *pyæmia*, which, with meningitis, is perhaps the most common cause of death in chronic suppuration of the middle ear. Again, if, instead of repeated rigors you only find rigors at the commencement, and the temperature oscillating, you have most probably to deal with *sinus phlebitis*, that is, with infective inflammation of the lateral sinus, or that portion which is more correctly termed the sigmoid sinus. What occurs is this:—In the first place thrombosis is developed, then the thrombus breaks down under the action of pathogenic organisms. The condition extends through the internal jugular, leading to pulmonary pyæmia, to metastasis in various parts, and ultimately death from pyæmia, but all through the medium of an infective phlebitis of the sigmoid sinus. Further points would be tenderness behind the mastoid process; this is probably due to phlebitis in the occipital sinuses. Then there may be a swelling down the line of the internal jugular vein, but the swelling found there is generally due to enlarged glands. In point of fact the vein may be so small that it can hardly be found—quite as small as the pneumogastric nerve, the reason being that from the thrombosis taking place up above, there has been no stream of blood coming down the internal jugular, and it has been allowed to collapse; or it may be that thrombosis has taken place at a former period and the thrombus has contracted. In some patients who have died from chronic suppuration of the middle ear, one can barely feel the lateral sinus, big as it normally is, the fact being that the thrombosis has taken place perhaps at some previous period in the patient's life during some feverish attack, and has settled down.

3. Now suppose that, instead of the temperature oscillating, it is SUBNORMAL, usually preceded by a rise at the commencement of the trouble. If the patient is dull and stupid, and has a somewhat localized headache, and he gradually gets more

and more comatose until he becomes unconscious, with slow pulse and slow breathing, then you are almost certainly face to face with *abscess of the cerebrum or cerebellum*. Abscess in the cerebrum is located in the temporo-sphenoidal lobe, while one of the cerebellum would be in the lateral lobe. To make sure of their presence it is necessary to perform an exploratory operation, such as I shall describe in my next lecture, under the heading of treatment. There is a great possibility of mistaking some of these conditions for enteric fever or tubercular meningitis, and the converse error, if less likely, has also to be guarded against.

You may say that I have not mentioned optic neuritis as one of the symptoms of abscess of the cerebrum. The omission is explained by the fact that optic neuritis is very often present when abscess of the cerebrum is absent, and it is very often absent when the other exists, so that it is no guide in that respect. On the other hand, it is almost always present in cases of sinus phlebitis. The ophthalmic veins and cavernous sinuses communicate with one another and with the lateral sinus; it is hardly possible for phlebitis to take place in the lateral sinus without having such an effect through the veins as would cause optic neuritis, so that optic neuritis is a sign of considerable importance in regard to sinus phlebitis.

These are some of the most terrible affections which you may have to deal with, and I trust you will not feel satisfied with knowing no more about diagnosis than I have told you to-day. I think you will find that the plans I have given you here are a fairly safe guide in analyzing cases, but still more in helping you to look in the proper directions for sources of more precise and full information on the subject.

Otalgia. (*Med. and Surg. Reporter*):

R *Chloral-camphor. ... 5 parts
Glycerini ... 30 parts
Ol. Amygdal. Dulc. ... 10 parts

Insert a piece of cotton wool, on which some of the above solution has been dropped, in the affected ear.

* Made by rubbing together in a warm mortar equal parts of chloral hydrate and flowers of camphor.

A CLINICAL LECTURE

ON

ECLAMPSIA.

Delivered at University College Hospital, Sept. 19th, 1895.

BY

G. F. BLACKER, M.D., M.R.C.P., F.R.C.S.,

Assistant Obstetric Physician to University College Hospital.

GENTLEMEN,—Eclampsia may be defined as attacks of tonic and clonic convulsions, the reason for which is to be sought in the processes connected with pregnancy, labour, and child-bed. Under the term Eclampsia we do not of course include convulsions due to hysteria, epilepsy, or cerebral lesions, such as may occur in pregnant women. Eclampsia is not a common disease, the frequency of its occurrence being estimated at about one in every 500 cases of labour; but this is no doubt rather a high estimate, and you may attend many hundreds of confinements without meeting with a case. It sometimes happens that a number of cases occur in rapid succession, and attempts have been made to explain this occurrence by the accompanying climatic conditions.

Eclampsia attacks primiparæ more commonly than multiparæ, and rarely occurs before the sixth month of pregnancy, but it may be met with as early as the fourth month, or even in the sixth week; when the first fit occurs after delivery, as a rule it immediately follows on the completion of labour, but its onset may be delayed for two or even three weeks.

It is of great importance that you should be thoroughly familiar with the premonitory and threatening symptoms of eclampsia. They usually take the form of gastric or sensory disorders; giddiness, loss of memory, and unilateral headache may be complained of, or the pregnant woman may suffer from nausea, vomiting, severe epigastric pain, failure of sight, and even complete blindness. If any such symptoms arise in a patient in the later months of pregnancy, you must at once make a careful search for œdema of the limbs or face, and carefully examine the urine, when the presence of albumen or casts may confirm your suspicions. And here let me say that I would advise you to make it a rule always to examine the urine, at least once before the

onset of labour, of every patient whom you are expecting to attend in her confinement.

Commonly you have no opportunity of observing the premonitory symptoms, and of trying to avert the threatened danger, since you are first called to the patient upon the occurrence of a fit. The characters of the fits themselves I propose to say very little about; they almost exactly resemble epileptic convulsions, only the cry is as a rule lacking, and the distinction between the tonic and the clonic stages is less well marked.

During the convulsions the respiratory muscles are involved, and respiration is arrested. As a result of this the patient becomes greatly cyanosed, and may even die of asphyxia. At the same time the temperature is often markedly raised, the pulse is small and intermittent, and involuntary escape of the contents of the bladder and rectum occurs. As the convulsions subside, the patient, who during the occurrence of the attack is entirely unconscious, in slight cases regains consciousness, or falls asleep, but in more severe cases remains quite unconscious, and even comatose. The attack usually lasts ten to thirty seconds, rarely as long as a minute. They vary in number from one to eighty, but as many as 160 have been recorded as occurring in one case. When the first fits occur during pregnancy, they are as a rule very quickly followed by contractions of the uterus, and the onset of labour.

The condition of the urine is of great importance; in some cases the amount passed is normal, more commonly it is markedly diminished, and on examination is found to contain albumen, often in sufficient quantities to cause it to become solid on boiling. Under the microscope you may find renal epithelium, blood corpuscles, fibrinous and hyaline casts, and crystals of urates and phosphates. There may, however, be no blood or casts, or the latter may be found in very small numbers. Sometimes no trace of albumen can be detected, but on repeated examinations you will find albumen present sooner or later in the large majority of cases. The transitoriness of its occurrence, and the large amount in which it is found are special features of the albuminuria of eclampsia. The amount of urea is in most cases diminished, both relatively and absolutely.

This complication of labour is one of great peril both to the mother and to the child; some

estimate of the mother's danger can be formed by the time at which the convulsions first begin; but the time of their onset makes very little difference to the child's danger.

When the fits first occur before the commencement of labour, the danger to the mother is much greater than when they occur first during labour, or after delivery. About twenty-five to thirty per cent. of the mothers die, while at least fifty per cent. of the children, the greater number of asphyxia.

The maternal deaths are due to asphyxia or exhaustion, and occasionally to cerebral hæmorrhage. But eclampsia does not alone expose the mother to the risk of losing her life, it further predisposes her to the dangers of post-partum hæmorrhage, septicæmia, and puerperal insanity—as all these conditions more frequently follow cases of eclampsia than normal pregnancies.

We must now pass to the most difficult, but the most interesting part of our subject, viz., the pathology of eclampsia. Our first knowledge on the point may be said to date back to the year 1843, when Lever pointed out that the urine of eclamptic women usually contained albumen. In 1851 Frerichs called attention to the very close relation that appeared to exist between eclampsia and uræmia. Two main views are held on the subject at the present time: one, that eclampsia is always the result of renal disease; the other, that while renal disease frequently accompanies eclampsia, yet it is not its cause.

The upholders of the second view argue that eclampsia is not the result of the albuminuria, because it can occur without any albumen being present in the urine; further, it does not occur in cases of chronic Bright's disease, and at post-mortem examinations cases of eclampsia frequently present no definite renal lesions. While we must admit that eclampsia does occasionally occur without albuminuria, yet in at least ninety-eight per cent. of the cases albumen is found, and even when no albumen is present in the urine this is no proof that the kidney functions are perfect. In support of this I may mention to you two cases recorded by Bouchard, where there was no trace of albumen in the urine, yet at the autopsy well marked amyloid degeneration of both kidneys was present. One must further always bear in mind the transitoriness of the albuminuria in these cases.

The second contention, that eclampsia does not occur in cases of chronic Bright's disease, is quite true, but here again we have no direct proof of the condition of the functions of the kidneys, and if they are deficient, the poisonous matters that should be excreted by them are absorbed into the blood in very small quantities at any one time, while we have every reason to believe that eclampsia is due to the sudden entrance into the blood of a considerable quantity of some kind of poison.

The third argument that no renal lesion is present is of little importance if the cause of the renal disturbance be a vasomotor one, as is not unlikely, since in such cases we would not expect to find any marked post-mortem changes. The pathological changes in the kidneys of eclamptic patients may be very varied. They may present all the appearances of the kidneys of acute or chronic nephritis, or a condition of hydronephrosis may be present with dilatation of the ureters. In some cases under the microscope acute degenerative changes are found such as are seen in the kidneys of acute septicæmia.

Some interesting experiments have been carried out on the blood of eclamptic patients with a view to showing the presence of a poison. It has been found that if 10 c.c. of healthy blood serum per kilogramme of body weight be injected into a rabbit, the animal dies; while it requires only 3—4 c.c. of the blood serum of an eclamptic patient per kilogramme of body weight to produce the same result. The toxic properties of the urine of patients suffering from eclampsia are also less than those of the urine of healthy people. We may, I think, take it as universally admitted that eclampsia is due to some poison in the blood; what the nature of the poison is we know not, we only know it is not urea. That the presence of the poison is due to some disturbance of the renal function, and that the pregnant uterus plays a part in producing such disturbance is also probable, but we are quite ignorant as to how or why the uterus produces such an effect in these cases. The sequence of events, at any rate in the cases where the kidney lesion is slight or absent, is no doubt as follows: The pregnant uterus, acting as the stimulus, produces some vasomotor disturbance of the kidney, this is followed by changes in the renal tissues, the presence of albumen in the urine, and some failure of the renal functions; as a result

poisonous materials accumulate in the blood, and these, by acting either directly upon the centres in the medulla, or by causing possibly a spasm of the arterioles of the body, and amongst others of those of the brain, produce the eclamptic convulsions.

We are very far as yet from understanding the true pathology of eclampsia, and this theory does not satisfactorily explain the cases in which all the signs of acute nephritis are present. Even if we admit that the kidney lesion is the cause of the presence of the toxic material in the blood, we have still no certain knowledge as to what it is that primarily produces the kidney lesion. Many ingenious theories have been put forward to explain the cases that occur without albuminuria. The best known of these is the Traube-Rosenstein theory; this view holds that as a result of the pregnancy the blood is in a watery or hydræmic state, and the arterial pressure is raised; these conditions together produce oedema of the brain, this in its turn leads to compression of the cerebral vessels, and resulting anæmia, and it is the anæmia that causes the convulsions.

In support of this theory the contention is brought forward that the blood of eclamptic women is especially hydræmic. The main objection to the view is the absence of any such cerebral anæmia at post-mortem examinations in cases of eclampsia.

There are two other views, which I need only briefly mention to you: the first holds that the liver, and not the kidneys, is at fault; that as a result of the failure of the functions of the liver, certain leucomaines which should be destroyed in that organ escape destruction, find their way into the general circulation, and give rise to the convulsions. Undoubtedly in some cases definite changes are found in the liver, but it has yet to be shown that these changes are the cause and not the result of the presence of the poisonous material. The second view is that the poison is due to a bacillus which has been found in the blood of eclamptic patients. This organism is said to enter the circulation through the uterus, in cases where endometritis has existed previously to pregnancy. The fact that eclampsia most frequently occurs in primiparæ, in whom endometritis is very uncommon, and the further facts that death of the child in utero, or artificial delivery, is frequently followed by cessation of the convulsions, a result

which would not occur were the fits due to the presence of an organism, are very strong arguments against any theory based upon a microbic origin of the poison.

We are driven back then to the, original view, that eclampsia is due to the presence of a poison in the blood, of the nature of which we are quite ignorant; that the presence of this poison is due, at any rate in the majority of cases, to a disturbance of the renal function, associated with definite changes in the kidneys; and further that the pregnant uterus plays a part, how or to what extent we know not, in the production of the kidney lesion.

In passing on to the treatment of eclampsia, one must first consider the treatment of albuminuria in pregnancy. In every case in which you find albumen in the urine of a pregnant woman, you should at once put your patient upon a strict milk diet. Diuretics and diaphoretics should be administered, and under the latter head one of the most valuable means of treatment is the hot bath. The patient may be put into a bath at the temperature of 100° F., the heat to be gradually raised to 104° to 106°; she is left in this for half an hour or an hour, at the end of that time she is packed in a sheet and blankets for three to four hours until profuse perspiration sets in. In place of the ordinary bath a hot-air bath may be arranged, and given at a temperature of 120° to 130° F. The baths may be repeated with advantage every day or every other day, and the bowels at the same time kept open with one of the hydragogue purgatives, such as compound jalap powder in 3j-3ij doses every two to three days, or one of the mineral aperient waters may be taken.

The most important agents in the treatment of the eclamptic convulsions themselves are chloroform and chloral. On the first occurrence of an attack, or of the restlessness which frequently precedes an attack, chloroform should be administered, and an enema of chloral hydrate of grs. xx-xxx given, to be repeated in an hour's time if required. Bromide of potassium may be combined with the chloral, but the latter drug in most cases appears to act better alone. The patient must, if possible, be at once put into a hot-air bath. A plethoric patient with a good deal of cyanosis may be bled to the extent of 20 to 30 ounces with advantage, but it is of questionable advantage to repeat the venesection when it fails to produce

any decided effect upon the attacks. In anæmic and weakly patients venesection should not be practised. If chloroform and chloral fail you, morphia is a means of treatment of great value. It must be given with caution to patients who are passing very little urine, but where a fair amount is passed morphia may be given by hypodermic injection with great success. You should commence with a dose of one-sixth of a grain, and carefully watch the result. Many obstetricians are in the habit of giving considerable doses of morphia, even to the extent of 2 to 3 grains in the twelve hours. By the use of morphia it is claimed that the mortality can be reduced to as low as 3 to 5 per cent. There is one drug you should not use, and that is pilocarpine; not only does it powerfully depress the heart, but by causing profuse bronchial secretion it greatly increases the risk of pulmonary oedema.

Shall you induce labour in cases of eclampsia? No definite rule can be laid down upon this point. The question of the actual induction of labour does not often arise, since, as we have seen, the first attack is quickly followed by uterine action. In the cases where your medicinal treatment has entirely failed, you should, if necessary, induce labour or assist its progress with a view to emptying the uterus as soon as possible. If the membranes are still intact, they may be ruptured with a sound, and dilatation of the cervix effected with Barnes's bags.

The patient must be kept deeply under chloroform while any such manipulations are carried out. After dilatation of the cervix forceps may be used or version performed. Rupture of the membranes is the best method of inducing labour in eclampsia, since it can be done with little disturbance to the patient, and it at once diminishes the tension of the uterus. Your aim should be to assist labour as far as possible, at the same time avoiding prolonged or unnecessary interference. The method of forcible delivery at any risk is bad treatment, and should never be attempted.

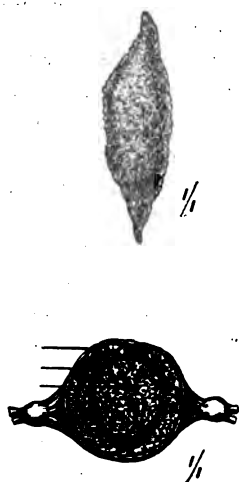
In the treatment of the convulsions occurring after delivery reliance is to be placed in chloral and morphia. Patients after delivery not unfrequently suffer from severe headache for some time; this may be treated by an ice-cap. You must on no account allow a patient who has had eclamptic convulsions to suckle her child.

ON A CENTRAL TUMOUR OF THE SPINAL CORD.

By J. BLAND SUTTON.

PRIMARY tumours either of the spinal cord or spinal column are rarities. It is a recognized clinical fact that a tumour arising within the cord disturbs the functions of the cord from its commencement; but as the nerve-substance appears to be elastic, and allows a great deal of gradual stretching without serious interference with its functions, a tumour may grow for a long time before it produces striking pathologic phenomena. On the other hand, when a tumour of the spinal column invades the neural canal it produces few signs until it presses the cord against the resisting walls of the canal, then the course of the disease is very rapid. The two following cases in a measure bear out these working rules.

This year Dr. Long kindly sent me from Maseru, Basutoland, the cervical portion of a spinal cord from a girl 14 years of age. About the level of the roots of the fifth cervical nerve, as near as could be judged, and lodged in the middle of the cord was a tumour of the size and shape of an olive (see fig.); the relation of the tumour to the proper tissue of the cord is shown in transverse section in the accompanying drawing.



Transverse section of the spinal cord, showing a central tumour. The upper figure shows the entire tumour.

In his very brief account of the case, Dr. Long writes:—"It is curious that this tumour caused no

symptoms until ten days before death: then there was rapid paraplegia."

What strikes one most is the circumstance that a tumour should attain such proportions in the cervical region of the cord before producing obvious signs. An examination of the cord above the tumour indicates that the fatal termination was due in all probability to hæmorrhage into the cord. The histologic features of the tumour are those common to a glioma. It is a fact worth notice that gliomata of the cord occur as discrete tumours, whereas in the cortex and great basal ganglia, crura, pons, and even the medulla they form diffuse masses indistinctly demarcated from the surrounding nerve-tissue.

In this case an operation was proposed by those in attendance, but the parents refused consent till the serious import of the signs was obvious even to untrained observers. Its uselessness was then clear, and even a cursory examination of the specimen shows that an attempt to remove it would have been a hopeless enterprise.

In connection with this case I will briefly relate the facts regarding a primary tumour of a vertebra in a horse. The animal had long been famous for its surefootedness: then, without obvious reason, it began to stumble when walking. In a few days it was noticed to fall when resting in the stable. A veterinary surgeon examined the horse and distinguished an irregularity on the left side of the third cervical vertebra. The horse rapidly became hopelessly paraplegic, and was killed. A transverse section of the third cervical vertebra revealed a spindle-celled sarcoma of the anterior articular process, which had encroached on the neural canal, compressed the spinal cord and reduced it to a narrow flattened band.

Lotion for Chapped Hands. (*Baelz. Med. chir. Centralb. Pharm. Post.*):

R	Caustic Potass...	...	$\frac{1}{2}$ gramme
	Glycerin		
	Alcohol	...	aa 20 grammes
	Aq. Distillat	...	60 grammes

M. F. lotio. Sig. Apply the lotion and rub it well in every 24 hours, after washing with warm water.

A CLINICAL LECTURE

GIVEN AT THE

BRISTOL ROYAL INFIRMARYBy HENRY WALDO, M.D. (Aberd.), M.R.C.P.
(Lond.),

Physician to the Infirmary.

GENTLEMEN,—The subject I have chosen for a clinical lecture to-day is a female patient, aged 16, with chorea, and in whom the jerky movements have been noticed for a fortnight. The family history is fairly good. There is no knowledge of insanity or epilepsy, but a sister of the patient has also had chorea. It is a disease very easily recognized by students, and you will have noticed that it occurs much oftener in girls than in boys. Unless these patients are asleep, they are quite unable to keep quiet. Their muscles, and especially those of the limbs, are in almost constant movement. If they attempt to perform any voluntary act, they cannot do it with precision. This condition has well been called "insanity of the muscles." In addition to these spontaneous movements and inco-ordination, their muscles are also very weak, and you have felt what a feeble grasp of the hand this patient has. This is usually the case; and in addition things are let fall in the occasional unintended relaxation of muscles, or there may be a delay of intended relaxation. The choreic movements and the paresis commenced on the right side; beginning in the leg, it quickly spread to the right arm, and it may at present be called a case of right hemi-chorea. There is seldom any affection of sensation in these cases; the only one this girl complains of is a tingling in the fingers of her right hand. Hughlings Jackson has remarked upon the frequency of motor aphasia in cases of chorea, and especially in right hemi-chorea. I have often noticed this, but our patient is up to the present an exception in this respect, as she has no loss of speech, the only thing noticeable about it being that it is rather hurried. Neither can I say there is any mental failure. The charge nurse tells me the patient is very intelligent, and that she considers her rather witty. But you must remember that this is up to the present by no means a severe form of chorea, and also that it is in an early stage. Some time ago I had a case in the same ward

whose intelligence was very defective. Beginning with mental dulness, inattention and forgetfulness, she went on to imbecility, and was speechless; but after many months' illness she quite recovered. I may also remind you that these patients are sometimes noisy, and even maniacal. There is nothing of importance in the respiration, although in some cases it becomes disordered in rhythm. Neither is there any difficulty of swallowing in this case. The temperature is normal, and it usually is so unless the patients knock themselves about a great deal, and so produce bed-sores. The electric irritability of the muscles and nerves are much the same as in healthy children. I am now going to say something of a very important sign, or rather an absence of it, in this case, and that is that there is no audible cardiac murmur present. You must not conclude from this fact that there are no vegetations upon the mitral cusps, for vegetations are found on the cardiac valves in all fatal cases, almost without exception.

There is undoubtedly a real connection between chorea and rheumatism, for endocarditis is scarcely ever seen *post mortem* unless the subject has been rheumatic or choreic. There have been many explanations as to the cause of the frequent mitral murmur in chorea. These patients are very often anæmic, and this has been considered to be the cause, and it has been spoken of as a hæmic murmur. It has been thought to be functional, whatever that may mean. And it has been very plausibly accounted for by supposing that choreic spasm of the muscoli papillares occurs and interferes with the due closing of the mitral valve. But the current view, and the most likely, is that the endocarditis is as much the cause of the murmur in chorea as it is in rheumatism. It is evident that the nervous system is primarily affected in chorea. The spinal cord has been selected by some observers, but the fact that choreic movements cease during sleep, the very time when the functions of the spinal cord are in a state of increased activity, is alone enough to throw very great doubt upon this theory. The ganglia at the base of the brain have also been thought to be the initiators. But an affection of that part of the brain which presides over the nerves and muscles involved in this disease could alone account for their disordered action, and so a disturbed nutrition of the nerve cells in the cortex will

explain the spontaneous movements, the inco-ordination, and the paresis, as well as the mental failure. The origin of this disturbed nutrition of the cortical nerve cells has been put down to embolism. The objection to the embolic theory is that these vegetations are firmly adherent, and that we do not find other organs, viz. kidneys and spleen, with similar anatomical peculiarities, participate in the process. The one condition of all others in which embolism, oftenest occurs is in mitral stenosis, and then I do not think vegetations form the embolus, but owing to the extreme stasis of the stream the blood clots, and embolism results. And then again, when cerebral embolism does occur, chorea does not develop.

It is thought now that the cortical nerve cells are poisoned in some way. This idea occurred to Osler some years ago, but it has only recently been taken up by the profession as a very probable theory. The poison may be a chemical one, or it may be a product of a micro-organism—a toxin. As I have before mentioned, chorea and rheumatism are very intimately related, and Dr. Newsholme believes that all the varied phenomena of rheumatism are suggestive of an infectious origin, although the specific organism has not yet been discovered, and he is anxious to abolish the term acute rheumatism, as he maintains that clinical, pathological and therapeutic considerations were strongly in favour of the view that rheumatic fever is a specific febrile disease.

I find that the knee jerks in our patient are normal, but in some other cases they are absent, and this is thought to strengthen the theory of a blood poison as in diphtheria. I believe imitation chorea is hysterical, and quite distinct from chorea as you see it in this patient. The onset of chorea is frequently connected with fright. This occurs, and usually the characteristic symptoms arise in about a week. This functional disturbance may be the exciting cause, the poison selecting the nerve cells when they are least able to resist it.

In treating a case of chorea, it must be borne in mind that the disease subsides of itself after eight or twelve weeks, under whatever treatment. Unless the case is very slight (these patients are usually benefited by gymnastic and other exercises), rest in bed—using a water-bed if necessary, and perhaps a board on each side—with careful nursing and abundant feeding, as they often waste much

and rapidly, is essential. It may be necessary to give chloroform in severe cases, but chloral with the bromides, or chloralamide or trional, given freely, is usually sufficient. Hughlings Jackson emphasizes liberal feeding and rather large quantities of brandy. Alcohol is no doubt more a sedative than a stimulant, and thus seems to do good. My patient is taking ten grains of phenazone in an ounce of water every four hours, if awake, as suggested by McCall Anderson, of Glasgow. She is also given cod-liver oil, which is one of the best restoratives for nerve cells or fibres.

We do not know an antidote to the imaginary poison. We assume that salicylates, in controlling the symptoms of rheumatism, does so by acting upon a micro-organism, but the same drug would not necessarily influence the toxine of that microbe. Arsenic has a great reputation in chorea, and as a nerve tonic no doubt does good—it, at any rate, lessens the anæmia—but the chief aim should be to treat the patient until the effect of the blood poison, or whatever it is, passes off. Before long there is good reason to hope that we may possess an anti-toxin for cases of chorea. The condition of the bladder should be attended to, as it is sometimes necessary to pass a catheter; and I have known incontinence of urine and fæces to continue for six weeks in a case of chorea.

Clifton, Bristol.

October, 1895.

REVIEW.

Dental Materia Medica. By Mr. J. STOCKEN.
Pp. 50. Lewis.

Price 4s.

This is a capital little book for those who habitually are treating carious teeth, and even for practitioners who only occasionally are called upon to treat toothache. We think that many teeth may be saved if general practitioners are aware of how much may be done by applications of remedies other than the extraction forceps. Several useful formulæ for tooth powders are included. The little book is now appearing in its fourth edition, and we can fully recommend it.

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 6, 1895.

TWO LECTURES ON CARIES OF THE SPINE.

Delivered at the National Hospital for the Paralyzed and Epileptic, Queen Square, London, July 3rd, 1895.

By W. R. GOWERS, M. D., F. R. S.,

Physician to the Hospital, and Consulting Physician to University College Hospital.

LECTURE I.

GENTLEMEN,—I show you to-day two cases, which illustrate very forcibly the way in which Nature not only ignores our distinctions, but compels us to disregard in practice those we have adopted even for practical ends.

The two patients before you are suffering from a disease which, in any other part of the body, would bring them under the sole care of the surgeon. Even in the part in which it has occurred, it would bring them under the care of a surgeon but for the accident that the part is adjacent to one region of the central nervous system. They are cases of caries of the spine. You are here in a "special" hospital, a hospital devoted to diseases of the nervous system, but if you go through the wards, about half the cases you will see are not cases of disease of the nervous system in the strict sense of the word. The nervous system consists of the nerve elements—cells and fibres, and of the supporting neuroglia, which is a persistent tissue, persistent from the embryonal tissue out of which the nerve structures have been developed. But this nervous system is contiguous to structures of very different nature; to elements of the vascular system, to parts of the osseous system, and contiguous to other structures, disease of any of which is liable to affect the nervous elements. The "disease" is that of the structures primarily concerned, but as soon as the nerve tissues are involved, it is regarded as coming into the class of diseases of the nervous system. Hence it is that all the practical considerations regarding a large class of the diseases

which are relegated to the care of those who concern themselves specially with the nervous system are diseases wholly different from the primary diseases of that system. A salient illustration of this is afforded by the cases before you. They are also an illustration of the wide range of specialism. True specialism is never narrow. It is well that all parts of our work should be thus linked together; that it should be impossible for any man to treat any case adequately unless he be versed in the recognition and treatment of all cases. While specialism is so far a necessity that without it our present knowledge could not yet have been reached,—of all the harmful things to those for whom we live our lives and do our work, the most harmful is exclusivism, in which a man has a knowledge of one class of diseases only. These considerations are illustrated in a curious degree by caries of the spine.

What is it? To put its pathology in a rough way, from the position of an outside view of surgical pathology, from the position of one who has only that knowledge which every one must have, or should have, to deal with any class of diseases, the affection may, I think, be thus described:—By some means, tubercular organisms are in the body of the patient. They may be purely acquired; for instance, from the milk of a tubercular cow. I saw, lately, a case of tubercular meningitis which seemed due to the milk of a tubercular cow, in one of those most perilous health resorts—a country vicarage. But the presence of tubercular organisms may be the result of heredity, and commonly is, though in what way, we have, I think, at present no conception. The most plausible explanation which we can conceive is that heredity involves some condition of the system—perhaps some chemical condition—whereby tubercular organisms, which are, perhaps, flying about for ever among us, are able to live, instead of being destroyed. It may be a peculiar condition of deficient energy on the part of the destroyers of organisms—the white blood corpuscles, whereby they are not able to appropriate and destroy the organisms which reach them. The apparent contradiction between the certain facts of inherited

tendency to tubercle, and the certain fact of the influence of the organisms, is one for which some explanation must exist. It is of no use to deny one set of facts because you cannot reconcile them to another set. Discern what the irreconcilable facts are—discern that they are facts, and if you cannot reconcile them, admit them, and trust to the future. You will not trust in vain.

When the organisms of tubercle effect an entrance into the body and thrive, they may thrive all over the system, or they may thrive locally. When they thrive locally, we have been accustomed to term the affection "scrofula"; when they thrive generally, "tuberculosis." They often do both. They often thrive at one spot only; that is so in many cases, though not in all, of caries of the spine. As far as we can discern, what happens is this:—From some cause local inflammation is excited in the spine; it may be of the slightest degree. Commonly, perhaps, it is a strain on the inter-vertebral substance which excites inflammation in it, because caries of the bone has been traced, in a large proportion of the cases, to initial processes in the inter-vertebral substance. But the cause may be exposure to cold, which, for some reason, has a local action. The process of local inflammation, slight as it may be, even so slight as to give rise to no symptoms, or only transient symptoms, seems to promote the local development of the organisms that may be in the system, and able to accumulate at the spot. Whenever and wherever organisms develop, there is about them a process of a secondary inflammatory nature, which further promotes their development. You find vessels developed, leucocytes extruding from the vessels, new tissue developed, such as we are accustomed to call inflammatory; and in all this there are local conditions favourable for the further growth of the organisms. There is a quantity of new tissue mixed with the organisms, not of great vitality, but the growth of which is at the expense of the proper structure of the part. The proper tissue wastes, so that in this growth of inflammatory tissue and organisms there is wasting of the bone tissue if the bone is involved. The condition progresses, and with it there occurs an effect which is a peculiar characteristic of the process of tubercular inflammatory growth—obliteration of the vessels. This obliteration cuts off the blood supply to some of the newly formed

tissue, and causes local necrosis. It causes an acute degeneration of the elements of tissue, whereby, instead of being continuous structures, they are broken up into minute fragments, which, from the lowered chemical changes they undergo, become fatty granulations and globules. These are mixed with leucocytes, or the products of leucocytal multiplication, in proportion to the acuteness of the process. The result is that this new tissue forms the peculiar material which, from its aspect, is either called caseous or is termed pus. Pus is produced when much liquid is formed to separate these minute particles; cheesy material when there is but little liquid. This caseous material, however, has little firmness; it cannot support the bone of the spine under the pressure to which the spine is subjected. Some of the bone gives way; and that which remains is insufficient to bear pressure, therefore this gives way also. The result is the crushing together of the bodies of the vertebræ, which causes the deformity. With the crushing together of the bodies of the vertebræ is a change in the canal, and the cord, where the course of the canal is abruptly altered, may by that means be compressed. But the new tissue produced by the tubercular inflammation has a larger bulk than the bone which it replaces, and the consequent swelling of the bodies of the vertebræ may alone compress the cord. Hence we have two classes of cases: first, one in which spinal-cord symptoms develop simultaneously with curvature; and another in which they develop before curvature occurs, and sometimes disappear when curvature occurs. For the curvature to occur, the mass of caseous tissue must be displaced, and if that mass has been pressing on the cord, curvature may actually relieve the cord from compression, and paraplegia, as I have known, may pass away as curvature develops. But in the great majority of cases neither of these mechanisms is that by which the cord is damaged. The common mechanism is that inflammatory products extend beyond the bodies, outside the dura mater, surround the cord, and compress it. At the level of the chief change, there is an accumulation of caseous material coming from the affected bodies, augmented, perhaps, by inflammatory and tubercular processes in the extra-dural tissue; this accumulates to such a degree as to compress the

cord. This is the common mechanism, with or without curvature, and it is a mechanism of extreme practical importance.

One other fact I must mention. Compression of the cord always involves tissue changes within it, involves these of necessity. The cord cannot be lessened in size to any degree without there being changes in structure, and those changes are of a more or less inflammatory character; there are vascular changes, and leucocytal accumulation; when these are rapid there is a considerable new formation of tissue, with destructive change in the nerve elements—a condition termed compression-myelitis. When compression is rapid, the inflammatory process is rapid. For some reason, which we do not yet well understand, the inflammatory process is sometimes rapid when the compression is slight. It is frequently so when the cause is malignant disease of the bones. It is remarkable that in cancer, in which you would expect inflammation to play the least part, there is, more frequently than in any other condition of compression, rapid inflammation of the cord, even when the pressure is slight. We meet with the same difference in caries; the change in the cord may be in proportion to, or in excess of, the compression. The difference is indicated by the rapidity with which the functions of the cord are affected. If this affection is rapid, it shows an excess of myelitis, provided the affection does not distinctly coincide with a cause of rapid pressure, such as a sudden change in the position of the bones. In one case I have recorded, a woman, with caries of the spine (which probably had just reached a crucial point, at which the collapse of the bodies was on the verge of occurring), sneezed three times, and the sneezing had the effect of developing curvature and paraplegia. In such a case we may assume that the compression was due to a sudden change of position of the bones, not to rapid inflammation. But if no ascertainable change of condition of the bones has been noted and there is rapid onset of paraplegia, you are justified in concluding that the cause is inflammation of the cord which has been set up in excess of its cause, alike in character and in rapidity.

Let me now call your attention to the facts of the two cases. I do not propose to give you any systematic account of the affection, because these meetings can be more usefully devoted

to considering the general features of disease in connection with the salient features of the cases that illustrate it.

This boy is aged 15 years, and is an example of the cases in which there is no family history of phthisis. His malady is apparently acquired tuberculosis, perhaps predisposed to by deficient general health. Remember what I said about our white corpuscles being the great destroyers of the microbial enemies to our life and health. Every part of our body, every structure, every tissue, shares the general health. You know that when an acute illness of a week or two varies the usual even tenour of health, it is revealed by a cross furrow upon the finger nails, because the cells of the epidermis, at that time, shared the general defective vitality. Therefore we can well understand that, in general defective health, the white corpuscles may be inadequate to destroy or digest the organisms which enter the blood. Hence, we can also conceive that previous defective health may dispose to the acquisition of tuberculosis.

A year ago, it was noticed that this boy was getting "round-shouldered," and, as expressed by his friends, "a lump was found in his back." He had no previous symptoms in his legs. He was advised to lie flat on a broad board for a certain period each day. Last Christmas he suffered from a severe cramp-like pain across the shoulders. In February the same pain returned across the stomach, and in a small degree in the lower part of the back. During the next few weeks, the legs became rapidly weaker, but he had no pain in the region of the curvature. He has now a well-marked prominence in the dorsal vertebræ; the spine below presents a curve forwards for three vertebræ, when it suddenly resumes its normal direction. Such a curve as that means a collapse of the bodies of the vertebræ whose spines are separated. In the case of the dorsal spines, you will observe that if the points are separated, it must be from a collapse of the bodies; but in every case it is necessary to consider carefully the exact direction of every spine, in order to endeavour to estimate the condition of the bodies, and the point at which they are chiefly crushed together. There is a curious deficiency in the illustrations of surgical pathology. There is an inadequate number of illustrations of vertical sections of carious spines, so as to afford practice in the habit of inferring,

from the position of the spines, the condition of the bodies. The importance of that is the greater on account of the surgical needs, which are likely to develop more and more.

Note that the boy has had no pain in his spine at the seat of the curvature, but has had pain in the chest and stomach. I cannot impress that fact too strongly upon you. Caries of the spine frequently gives rise to spinal pain, but it not seldom gives rise to no pain there, though it seldom fails to give rise to some extrinsic pain. When you find a change in the bodies of the vertebræ, such as to cause a great difference in the relations of the spinous processes, there must be a disturbed relation between all parts of the vertebræ involved; and the nerve roots may be compressed as they pass through the intervertebral foraminæ. The inflammatory changes which extend from the bodies to the structures outside the dura mater, and to this membrane itself, and sometimes within it, cannot fail to affect the nerve roots. Their affection seems seldom to give rise, in the dorsal region, to muscular symptoms, but it often gives rise to sensory symptoms. The reason why the motor symptoms are commonly absent is, apparently, because the muscles of the trunk are supplied by so many nerve roots, that damage to a few of them seldom causes recognizable motor symptoms. But irritation of the sensory roots causes extrinsic pain to be one of the symptoms of the disease of greatest diagnostic importance. In the arms there is not the same relation of the muscles to a series of spinal nerves that there is in the dorsal region. Caries of the cervical spine may be revealed by conspicuous muscular atrophy. In the dorsal region pains alone are the symptoms of irritation of the nerves, and of damage to them. In the other patient you will see a curvature somewhat similar, except that its upper part presents a less sudden prominence. If you exclude the suddenness of the upper part of the curve, the two are not dissimilar.

It is common, at least it is frequent, for such pains to precede any recognizable symptoms of disease of the bones. Cases of caries of the spine are often mistaken for intercostal neuralgia. I remember one case of unquestionable caries of the lower dorsal region which, for about nine months, had been treated as a case of renal colic. This point is especially important, because in the early

stage of the affection there may be no curvature, no spinal pain, and even no tenderness.

Each of these patients has come into the hospital on account of paraplegia; the man was here for the same symptoms in 1891. In him, the first symptom was pain in the back after sitting long at a stretch, and about a year later he noticed some prominence in the spine or about it. Shortly after noticing that prominence, his legs became weak; weakness and stiffness soon involved the whole of both legs, until he had to lie. Then he noticed some numbness in the feet, and came to this hospital as a patient in February, 1891, unable to move his legs, and unable to feel up to the level of the umbilicus. Sensory impairment from caries is much less common than motor impairment. It is a rule with the nerves and cord alike, that an apparently equal affection of all the structures has far more effect on the motor structures than on the sensory. We do not know why. It may be simply that the impulses which can pass a given obstruction are able to cause adequate sensation, and yet are unable to influence the muscles. Although we do not know the explanation, the fact is important. The sensory impairment in this man shows that the damage to the cord must then have been extreme. He entered the hospital in February—at Christmas he went out able to walk seven miles without fatigue, with perfect sensation. He continued well until last October, when his legs again became weak, and he came in the second time at the end of January. At that time he could not raise his legs from the bed. He could adduct them very feebly, but could not flex either knee, and could not flex his ankles. He is gaining power, but slowly; power is always more slowly regained in a second attack than in a first. In him, movement is also prevented by the spastic state of the limb, which always results from disease of the dorsal cord, in consequence of the secondary degeneration of the pyramidal tracts. But this spastic state is capable of passing away completely if the pressure on the cord is removed. There is no degree of spastic paralysis that may not be recovered from if its cause can be removed before there is total destruction. I may, on another occasion, be able to show you this patient in a better state.

In the boy, as I have said, the first symptom was curvature, followed by pain. He came in

on March 20th, when he had very little power in his legs. He could then draw the legs up, but could not raise them from the bed. He has gained power considerably, as you see.

In each case there was a very conspicuous degree, not only of muscle reflex action, but of the superficial reflex action. To this I would draw your attention, but I must postpone until our next meeting the consideration of some other practical points. Remember that excess of the superficial reflex action is a common symptom in cases of compression of the cord, as distinguished from damage to the cord at a similar level by a primary morbid process within it. A great excess of superficial reflex action in the legs should always attract your attention, and you will find that in some cases it is a trustworthy indication in the diagnosis between organic and functional disease.

A CLINICAL LECTURE

ON

ORGANIC STRICTURE OF THE URETHRA.

By A. G. MILLER, M.D., F.R.C.S.E.,

Surgeon to the Royal Infirmary, Edinburgh.

Lecturer on Clinical Surgery; Examiner in Surgery and Clinical Surgery, Royal College of Surgeons, Edinburgh.

GENTLEMEN,—The causes of organic stricture of the urethra are two: gonorrhœa, and injury. I shall not refer to spasm or to simple inflammatory swelling as causes of stricture, because I do not consider that they produce stricture. They may cause retention of urine, but not stricture. Please do not confound the two. They are essentially different conditions.

I shall not take up injury as a cause at this time. Traumatic stricture will be considered some other day. Gonorrhœa is the most common cause of stricture, and is quite sufficient to occupy us to-day. I shall not even discuss the pathology of gonorrhœal stricture; I shall content myself with the statement that stricture follows gonorrhœa, and that it does so more frequently in cases that are badly treated, or not treated at all.

Taking for granted then that stricture of the

urethra is a result of gonorrhœa, let us consider what are the symptoms.

2. The patients complain of difficulty in making water, which comes in a small stream. There is a past history of gonorrhœa, of alcoholism, and of retention. But these are not the first symptoms of stricture. Let us see what these are.

We have seen that the cause is a badly treated or neglected gonorrhœa. The copious purulent discharge characteristic of infective urethritis has been followed by a chronic discharge—a gleet. This slight discharge may be counted the first symptoms therefore of stricture, because it means irritation of the urethra, irritation that may cause contraction. Another early symptom is frequency in making water—this also being the result of irritation. These two symptoms may be present for a long time before any perceptible contraction of the canal takes place. Perhaps therefore they should be called premonitory symptoms. I describe them to you because patients seldom attach importance to them, and yet they are very important indications of the existence of an irritation which very probably will cause a stricture. Given then a slight discharge—a drop or two in the morning—with an increase in the frequency of micturition (the patient having to retire every two or three hours), a suspicion of commencing stricture should be aroused, and the urethra should be carefully examined in the manner that I will describe immediately. When a constriction of the canal has fairly formed, then the symptoms become distinctly marked. There is frequency in making water, or, as the patient generally puts it, difficulty in retaining water—most marked in cold weather, and after indulgence in alcoholics. You will find as we go on that alcohol has a great deal to do with stricture, and is perfect poison to anyone suffering from that affection. All the bad cases of stricture that I have had to treat have been chronic and excessive drinkers.

But to continue. In addition to the patient having to make water frequently (say every two hours), he has some difficulty in passing it. He has to press a little, and takes longer than he used to do. Moreover when he has, as he thinks, finished the performance, or has got tired of squeezing, he finds that a few drops come away into his clothes. These few drops increase in quantity as time goes on, and one may, from this

symptom alone, frequently diagnose an old stricture case, from the urinous odour which pervades his garments.

I have said nothing about double streams and forked streams, because these so-called symptoms may be present temporarily in persons who have no stricture, and are often absent in well marked cases. Now let us consider what is the state of matters inside the urinary passage of a person who has the above symptoms. First there will be a contraction (much more frequently several contractions) in the lumen of the urethra. There will also be some hypertrophy (compensatory) of the bladder to overcome this obstruction. Also there will be some (residual) urine, left in the bladder at the close of micturition, at first a few drops, but by and by some ounces.

How does this urine come to be left in the bladder? I think that the simplest and most reasonable explanation is that, micturition being difficult, the patient gives up the effort to pass his water before his bladder is quite empty. He has got relief and is quite contented with that. Then in course of time, for the process is slow, and this symptom of stricture appears only after the stricture has existed for a long time, years probably—in course of time the bladder, never being perfectly emptied, becomes accustomed and adapted to the retention of a certain amount of urine, and, the quantity increasing, the bladder becomes dilated, even though it may be at the same time somewhat hypertrophied. The damming back and dilatation process may extend further back, namely to the ureters and kidneys. How this occurs is more difficult to explain. I cannot say that I have seen it, but it is described.

There are many other changes that may take place, and pathological conditions that may appear in connection with stricture, but these I prefer to consider as complications, and will refer to some of them afterwards.

3. At present I will take up the question of diagnosis. Not that stricture, as a rule, requires to be diagnosed from other affections, but because an exact diagnosis of the condition of the stricture is necessary before treatment can be properly decided upon and undertaken.

There are several things that the surgeon must make out—first the existence of a stricture, then the situation, the number of strictures, and the

essential character of each. The existence of complications and other affections must be made out also.

The stricture is best diagnosed by the use of a flexible bougie with a large head. Such an instrument of course is easily arrested by any obstacle. In this way commencing strictures are most easily discovered. Rigid and heavy instruments will pass readily through obstructions that will completely stop a flexible and large-headed one.

Let me suppose then that you employ such an instrument—say No. 15 English—it is arrested in its passage along the urethra about half an inch from the external orifice. You take a smaller one therefore—No. 12—and it goes further down, say two inches, and is there obstructed. In withdrawing it you find a slight catch where No. 15 was stopped. This is a very common experience, for it is in coming out of the urethra that the abrupt shoulder of an acorn-headed bougie catches a slight contraction most readily. You now take a smaller bougie, No. 9. It goes in a little further and is then stopped. You may take a smaller still, say No. 6. It goes further, but is completely stopped at $4\frac{1}{2}$ inches. You may now lay aside these instruments. They have told you what you wanted to know, viz., that there are, not one, but several strictures, of varying diameter, the tightest and firmest being somewhere about the bulb.

This I am giving you as a typical, not a rare case, as you may perhaps think. In gonorrhoeal stricture the rule—at least in my experience—is that there are several strictures, and they seem to increase in tightness the further from the orifice they are. Why the older surgeons so constantly wrote about stricture of the urethra as if there were only one strictured point, was due I suppose to their using rigid instruments for diagnostic purposes, which, as I have pointed out to you, is a mistake. Besides, the older surgeons seemed to be contented with the discovery of the tightest stricture, and with the treatment of it.

You have now to ascertain the condition of the deepest and tightest stricture. For this purpose you employ firm metal bougies. The best are those designed by Sir Joseph Lister.

They are probe-pointed, and therefore pass more easily along the urethra than any other kind of instrument. They are also graduated or wedged

on the shaft for the dilatation of the stricture, as we shall see presently. Your object now is, not to discover strictures—that you have done already—but to find out the *largest* instrument that you can get into the bladder. You begin therefore with a No. 6 (the smallest you had tried of the flexible bougies), and take a smaller and smaller size till you get one through the stricture into the bladder. You now know what size of instrument the strictures will permit to pass. You will also have ascertained the character of the tightest one, whether soft or hard, rough or smooth, straight or crooked. You will also by this time have found out if there are any false passages.

To complete your diagnosis of the case, you will have to investigate further the condition of the bladder, kidneys, perineum, prostate, and the patient's general health, habits, etc.

4. Having thus completed the diagnosis, let us consider the treatment of an ordinary case—complications we must reserve for after consideration, if we have time. We have found out, let us suppose, that the patient has several constrictions, and that the tightest one, which is always near the bulb, permits the passage of a No. 2 Lister bougie. What are we to do further? That will depend on the characteristics of the stricture, whether it is soft and easily dilated, or hard and rigid, or what Mr. Syme use to call "resilient."

Let us take first—an easy case. You have passed a No. 2 Lister bougie, and shoved it on till the point has entered well into the bladder. You will remember that the bougie is No. 2 only at the bulbous point, the shaft being graduated to three sizes larger. If the instrument therefore has been pushed (never of course with violence) well into the stricture, it will have been dilated to size 4 or 5. You may therefore take a No. 4-7 Lister bougie and pass it, and perhaps another larger still—and then you will have done enough for the first *séance*.

Now I cannot here enter into all the manipulative details of passing instruments into the urethra, but I shall give you some rules which, from my experience, I can recommend to you.

1. Never use violence or be in a hurry. Hold your instrument lightly and use it carefully.

2. Never pass more than three instruments at a *séance*, and allow three days to intervene between each *séance*.

3. When you feel something give way or tear, and when you have caused bleeding, stop.

4. Have your instruments warm. To lubricate my instruments I use vaseline with 10 per cent. of eucalyptus and cocaine. Bougies may be very easily made warm and aseptic by passing them through the flame of a spirit lamp.

5. Administer a dose of quinine (gr. v) and morphia (gr. $\frac{1}{4}$) or Pot. Brom. (gr. x) either before or immediately after the operation (before is preferable), to prevent rigors.

In the treatment of a simple case, such as we have been supposing, the passing of two or three bougies twice a week may be carried on for some weeks, till you are able to pass a full-sized instrument—each time of course commencing with a larger-sized bougie than the time before. After you have got up to (say No. 15-18) the largest size that the patient's urethra will admit, you pass that instrument (which my father used to call the "protesting bougie") once a week, for a week or two, then once a fortnight, for a month or two, then once a month, and then once in three months, for a year or two. In this way cure can be perfected in a simple case, but the process takes a long time, as you see.

Now I must give you some warnings. You will seldom procure this easy course, and happy result, especially in hospital practice, because of two things mainly:—

1. The patients do not come regularly for treatment, or they cease altogether when the cure is only half completed. They get tired of the prolonged treatment, not realizing the importance of the "protesting bougie," which prevents recontraction, or shows at once should such recontraction commence; or they imagine, when they see their urine coming in a full stream, that they are quite cured, and that you are only wanting fees, or practice to keep your hand in.

2. Another thing that often prevents a cure being completed is indulgence in alcohol and venery. These seem always to irritate and aggravate strictures, as we shall see again in treating of complications.

Some surgeons will tell you that stricture of the urethra is incurable, and that no method of treatment is perfect. I would not dash your hopes in this way. I think that strictures are perfectly curable, and the methods of treatment are satis-

factory enough (till someone invents a royal road). The blame rests not with the methods, but with the patients, who are almost always impatient of treatment, and habitual indulgers in alcohol and other vice.

Let us now consider the treatment of a more difficult and troublesome type of case. You have persevered in the above described treatment for some time, and, though your progress has not been so rapid as you would like, you have persevered steadily and got on to No. 7-10, when one day you find that you cannot pass that instrument, and you have to try smaller ones, till you come down to 4-7 or 3-6. You start afresh from that point till you get up to where you were, or you may get on further (say to 9-12 or 10-13), when you will be thrown back again to smaller sizes. Now this may be due to indiscretion and folly on the part of the patient, or it may be the peculiarity of the stricture. In the former case the patient is to blame, and you must let him understand that he must bring his reason and resolution to assist you in the treatment, otherwise his stricture will never be cured.

If the nature of the stricture, and not the patient, be the cause of the recontraction, then you have got a "resilient," or elastic, or spasmodic stricture to deal with, and something must be done specially to meet and overcome this peculiarity. Antispasmodics are of no avail, in my experience, and therefore I do not think "spasmodic" is a good description of this kind of stricture. Without going into the pathology of this condition, I will merely mention the methods of treatment which I have found specially useful in such cases. These are three—rupture, internal urethrotomy, and external urethrotomy.

1. Rupture, of this "resilient" stricture, may be effected by means of Holt's instrument, or by passing Lister's bougies in rapid succession till the stricture is felt to give way.

2. Internal urethrotomy—cutting the stricture from within—has to be most carefully done, not because it is either a difficult or a dangerous operation, but because the stricture, on account of its elastic character, is very apt to escape, and not to be cut at all. I like to put my finger on the outside of the stricture, and to press against the urethrotome, and thus to make sure that the constriction is really severed.

3. External urethrotomy (Syme's operation) should be performed only when other means have failed. I shall refer to the operation again in connection with the next type of case.

This brings me to the third type of case, the hard, rigid, cartilaginous, and not unfrequently tortuous stricture. In such a case treatment by gradual dilatation is so slow that some other method is employed to "hurry up" the treatment. In some cases no progress whatever is made, and there is absolute necessity for some more effectual method of treatment. In such cases we may have a choice between the three methods mentioned already, internal urethrotomy, external urethrotomy, and rupture of the stricture. Of these I most frequently employ Syme's operation, because there is so much difficulty in passing the instruments necessary for the other methods. Syme's operation may be described shortly as follows: The staff, with its fine point, abrupt shoulder, and groove for the knife, is passed through the stricture up to the shoulder. The patient is then placed in the lithotomy position, and an incision made in the mesial line over the situation of the stricture. When the surgeon can define the stricture with the index finger of his left hand, he places the point of his knife in the groove of the staff *behind* the stricture and then cuts *forward*, dividing the stricture in its whole length. The staff should then be passed on into the bladder, to demonstrate that the way is clear. After the staff is withdrawn an S-shaped catheter is passed into the bladder from the perineal wound, and tied in. This catheter may be left in for two or three days, and then the passage of instruments by the external orifice of the urethra may be commenced. There are three mistakes apt to be made regarding this operation. 1. It is often called perineal section. Syme named it external urethrotomy. 2. The section of the stricture is said by some to be from before backwards. Syme made the section from behind forwards, and pointed out that the former method might fail from the tough stricture tissue being shoved back before the knife, and, therefore, not properly divided. 3. It is sometimes supposed that the operation is sufficient of itself to cure the stricture. This is not so. It is merely the commencement of the treatment which is to result in cure. The use of bougies after the operation to prevent recontraction

tion, and to maintain a patent urethra, is absolutely necessary. The urethrotomy merely gives the treatment a fair start, and a very fair start it is, for, instead of labouring away for months, and making little or no progress, one is able, or should be able, to pass a full-sized bougie at once; and, with proper care in the after treatment, one should never need to use a small instrument again. My plan is to commence with a 10-13 or 12-15 Lister bougie a few days after the operation, and to pass that instrument about twice a week till the perineal wound heals—which it generally does in three weeks. I then go on dilating till the largest possible instrument is reached, and then that one is used as the "protesting bougie" till cure is completed.

I now wish to say a few words in regard to a class of cases frequently called "impermeable" or "impassable" strictures. Of the type of cases so called I suppose that I have seen a good many examples, like all other surgeons, but I prefer to call them "difficult" cases, because, with care and perseverance, and the employment of proper instruments, the strictures should not prove impassable or impermeable. Before speaking of the treatment of these cases I must state that they are usually complicated with more or less complete retention of urine—fortunately not always. Retention I shall deal with as a complication afterwards, if we have time; at present I shall speak of the treatment of difficult and tight strictures.

The methods recommended are: external urethrotomy, internal urethrotomy, filiform bougies, and Wheelhouse's operation—besides some other performances. The last operation I have never performed, and therefore prefer to say nothing about it. The first operation I have just described, and would only add that I have frequently performed it in cases of difficult and tight strictures with most satisfactory results. If a small enough staff be employed, there are very few cases that should not succumb to careful and skilful manipulation. But remember that these cases require both care and skill, that is to say experience, for their successful management. It is not sufficient to say that there is no such thing as an impassable stricture, and then to force an instrument into the bladder somehow or another. Neither is it safe to think too much of Mr. Syme's oft-quoted (not unfrequently misquoted) dictum, that if urine can

pass out (and there is usually a slight escape of urine), an instrument ought to pass in.

Internal urethrotomy I have not tried in difficult strictures for obvious reasons. Fine, flexible, filiform bougies I have tried, but have never got any satisfaction from them. I much prefer the instrument which I adapted from Syme's staff, and which I have described as the Syme-Spence-Lister bougie.* The instrument is made of solid steel, and is consequently heavy and rigid, and therefore requires no force to make it pass down the urethra, and its exact position can always be ascertained. In these respects it is like a Syme's staff. The instrument has also got a very fine probe point, as employed by the late Prof. Spence, which enables it to "engage" the smallest strictures. Lastly, its haft is graduated or wedge-shaped, like the Lister bougies, which enables it to be used as a dilator, and a pioneer to other instruments of larger size. The rules that I give for the proper employment of this instrument are to use care and patience, and to let the instrument find its own way rather than to attempt to guide or push it.

5. I must now say a few words on the complications of stricture. They must be very few indeed. I can do little more than name them.

(a) Spasm not unfrequently complicates strictures, causing retention of urine. A common cause of spasm is alcoholic indulgence; another seems to be exposure to cold. The treatment is soothing remedies, such as a warm bath, morphia, potassium bromide, and rest in bed, with milk diet.

(b) Retention of urine may also occur, the result of spasm, as we have seen, and is always a serious and troublesome complication. Retention is sometimes spoken of as a symptom of stricture, but I think it is more correctly a complication. The subject is a wide one, and I cannot dwell on it at present; I will merely say that the means of relief are not limited to catheters. The warm bath and antispasmodics, referred to already, may relieve the patient and enable him to pass water. The passing of a bougie, which is generally an easier and safer thing to pass than a catheter, may enlarge the stricture sufficiently to let urine trickle away; and in desperate cases aspiration of the bladder may be practised safely, and will give great relief.

* See Edin. Med. Journ., Oct., 1893.

(c) Rupture of the urethra, with extravasation of urine, is a most serious complication, requiring immediate and important operative treatment. This subject I cannot enter upon farther than to say that external urethrotomy (Syme's) is an operation that generally suits such a case, for by it the stricture is thoroughly treated, as well as the extravasation.

(d) False passages not unfrequently occur as complications of stricture cases. They are more troublesome than dangerous. There are one or two things I would like to say about them.

1. They can be caused by large as well as by small instruments.

2. Their formation is generally indicated by a sudden jerk of the instrument, followed by a gush of blood.

3. They may heal; but often become established as cul-de-sacs, into which instruments have an unfortunate tendency to slip.

4. Old false passages differ from the normal one in two respects mainly. They are not in the mesial line generally (which can be ascertained by putting a finger in the rectum), and they are less sensitive than the urethra. In the case of a patient who has been long under treatment, and who has one or more false passages, when the instrument slips in quietly and easily, it is apt to be in the false passage, but when the patient winces, and the instrument is caught, the bougie is more likely to be in the stricture. N.B.—False passages go off from the urethra before one comes down to the stricture.

5. If the stricture becomes cured, and instruments are no longer passed into the false passages, the latter will probably close.

(e) Urinary fistulæ are not uncommon complications of old standing strictures. Fistulæ may be in the perineum (perineal fistula), or anterior to that situation (urethral fistula). They may be the result of rupture of the urethra, false passages, or urinary abscesses. They may be treated by various methods. I shall merely say here, that if you cure the stricture, the fistulæ generally close and heal up of themselves.

(f) Urinary abscesses are sometimes caused by stricture. They form in the cellular tissue, and are apt to result in fistula. The abscess usually opens into the urethra first, then on to the cutaneous surface, so forming the complete fistula.

Such abscesses should be opened as early as possible.

(g) Septic cystitis is not an uncommon complication of stricture, more especially if retention has occurred, and catheterism has been resorted to. It may disappear when the stricture is cured, or it may persist, and give rise to another and more serious complication, viz., septic kidney.

(h) Two more possible complications I shall merely name, prostatic enlargement and stone in the bladder.

I here hasten, in conclusion, to say a few words on the preparation of a patient for instrumentation, and the after treatment of the case, with a view to curing the stricture.

Not unfrequently a stricture case comes under our notice first with urgent symptoms of retention. This complication demands immediate attention, and no special preparation of the patient can be indulged in. But when we have time the patient ought to have a warm bath and a good wash, and ought to be put to bed and kept on milk diet for a day or two. In the meantime he should be carefully examined, his history and habits ascertained, the condition of his internal organs investigated, as well as the peculiarities of his stricture made out. It may be advisable also to administer some antispasmodic (as opium or potassium bromide) or antiseptic (as quinine, salol, or sulpho-carbonate of soda). Such precautions are advisable when they can be carried out, for by such means one may prevent high temperatures and other unpleasant consequences following the treatment of the stricture.

Regarding the after treatment of instrumentation, I have one or two rules to give you. Always administer a dose of quinine, with morphia or potassium bromide, after passing instruments, and place the patient in a warm bed if possible. In this way high temperatures may be avoided. If the temperature should rise, don't be afraid of it, unless it remain up. If it be due to the passing of the instrument, it will come down in a few hours.

Warn your patient against alcohol. You may generally prove to him, from his own experience, that alcoholics of all kinds do harm to his stricture. They oblige him to make water more often, cause him to strain more, and not unfrequently produce spasm with retention.

Lecture him also on the importance, the necessity of his returning regularly to have instruments passed, if there is to be a cure. I have already told you that such patients deprive themselves of the only chance of cure by neglecting to return regularly. They give many excuses, but the principal reason is that they are moral cowards. They are mostly old alcoholics (as I have already told you), and are in the habit of indulging themselves in all manner of excesses.

Don't suppose that because so many cases of stricture are not cured, but go from bad to worse, that therefore our methods are imperfect and insufficient. The methods are good enough if properly and thoroughly carried out.

CLINICAL REMARKS

ON

CASES OF HEART DISEASE,

In the wards of University College Hospital.

By F. T. ROBERTS, M.D., B.Sc., F.R.C.P.,

Physician to the Hospital.

It is not my intention on this occasion to discuss heart diseases systematically, but merely to indicate certain prominent points which the cases at present in the wards illustrate. I will go more fully into the consideration of this class of diseases at a future time.

Case 1.—This man was not originally a heart case, the cardiac affection being a result of renal disease and its consequences. It is, therefore, an example of heart mischief secondary to disease of some other organ or organs. There is hypertrophy, with some degree of dilatation of the left ventricle, the enlargement being due to difficulties in the circulation, and changes in the arteries, caused by the kidney trouble. There is an indistinct murmur at the apex of the heart, which indicates some degree of mitral regurgitation. Auscultation at the base reveals a very loud, accentuated, second aortic sound, which is not directly connected with the heart, but depends on increased arterial tension. The cardiac disease does not give rise to any definite symptoms, and is at present quite subordinate to the kidney affection; the patient does not even complain of shortness of breath.

Case 2.—This second case is one of very pronounced cardiac disease, which may be described as primary. There is nothing in the history to point to any definite cause of the mischief, though it is probably of rheumatic origin. There has been no rheumatic fever, and no sudden strain or violent and prolonged exertion to which we can attribute his condition. The patient only speaks of some indefinite rheumatic pains. I therefore remind you that you must be prepared for cases of pronounced heart disease of which you are unable to ascertain the cause, and you must not assume that there cannot be such disease because you can find nothing to

account for it. This patient has marked shortness of breath, and this is the symptom which brought him to the hospital. It is an important one in relation to cardiac disease, and should always be borne in mind in this connection. There has been slight cedema of the legs. The anæmic condition which you observe was much more marked a few days ago, though he is still somewhat pale. I wish to emphasize the fact that this is a very complex case of heart disease, and you will find that this is the state of things in a large proportion of instances where this organ is affected. Indeed cases which last a long time almost necessarily become more or less complicated. You can see at a glance that the cardiac impulse is far outside the nipple, and diffused over a wide area; and by placing the hand over the heart you can detect more than one thrill. Auscultation reveals various murmurs—double mitral, double aortic, and a doubtful tricuspid, indicating serious disease at these orifices. There is great enlargement of the organ, due to both hypertrophy and dilatation, which are more or less general, but affect the left cavities more than the right. A condition I always look for in chronic cases such as this is pericardial adhesion, but there are no signs of it here, and the heart-muscle is in good condition. You should take every opportunity of learning and studying murmurs in actual patients, instead of merely learning their description from books, and this case affords you excellent opportunities for doing so.

Case 3.—This boy illustrates heart trouble brought on by, and consequent upon other morbid conditions of the chest. He has emphysema in a pronounced form, some degree of bronchitis, asthma, and phthisis, and there is some reason to believe that he has limited pneumo-thorax. We can feel a marked impulse in the epigastrium, due partly to displacement of the heart in a downward direction by the pulmonary conditions; there is also a tendency to over-distension of the right side of the heart, which ultimately ends in permanent dilatation and tricuspid regurgitation. The obstruction of the circulation in the lungs necessarily acts upon the right side of the heart in these ways. Cases in which the tricuspid orifice becomes so large that the valves cannot close it, thus leading to obstruction of the general venous circulation and its consequences, are some of the most troublesome we have to deal with in practice. The obstruction is not very marked in this case at present, but it may become so, and lead to dropsy, cyanosis, and other phenomena. Probably the tricuspid valves are not quite competent, but there is no actual disease at this orifice, and the mitral and aortic orifices are quite normal.

Case 4.—The next is not a simple heart case by any means, and is altogether different from those we have thus far considered. The symptoms are very prominent, and such as at a superficial glance would seem to point to the heart as their chief cause. The patient is not merely short of breath, but he cannot lie down on account of his dyspnoea—so-called "orthopnoea"—and he has extensive dropsy, though that in the legs and scrotum has diminished somewhat since his admission into hospital. His liver is enlarged considerably; the abdominal walls are oedematous; and there is a certain amount of ascites. As a matter of fact this case is a very complicated one. There are marked changes affecting the chest walls, pleuræ, and lungs, all tending to embarrass respiration, and to obstruct the venous circulation. In addition, however, I believe that there are decided degenerative changes in the cardiac walls, as well as in the aorta and other arteries, and these are of great importance under existing conditions. No murmur can be detected, and there is no positive evidence of any valvular mischief. No doubt more or less dilatation is present. The tendency is to regard combined chest cases, presenting the symptoms now under observation, as merely cases of heart disease, and to ignore other factors which are in reality of great consequence. The cardiac signs in this instance are by no means pronounced, and have to be carefully sought for.

Case 5.—You will notice how child-like this girl looks, yet she is 17 years of age. The case is a striking illustration of the retarded growth and development not uncommonly asso-

ciated with early cardiac trouble. This patient has been in the hospital four times, and has had several definite attacks of rheumatic fever, which accounts for her heart disease. It does not belong to the congenital group. She is always troubled with her breathing, and cannot lie down comfortably, but there is only slight oedema. The case affords a good example of double mitral disease, both obstruction and regurgitation. A frequent morbid condition set up in children suffering from rheumatic fever is pericarditis, leading to pericardial adhesion, which is probably present here. Such patients often have endocarditis and pericarditis together. In this case, as you see, there is well marked hypertrophy, and the heart is doing its work very well under the circumstances.

Case 6.—The most striking feature observed in the next patient is that she is extremely anæmic, and this appearance may be a very prominent symptom of cardiac disease. Superficially investigated, this case appears to be a simple one, but I believe it is not, and that there is probably double mitral and aortic disease. The only history that can be obtained from this patient suggesting a rheumatic origin is that she had "growing pains." In doubtful cases which you cannot trace to rheumatism, remember always to ask about chorea, which is popularly known by the name "St. Vitus's dance," but in this instance there is no history of any such complaint. The prominent morbid condition in this case is mitral obstruction or stenosis, as evidenced by the very pronounced thrill at the apex, not merely presystolic, but prolonged almost throughout the whole diastole, and terminated by an abrupt and sharp impulse; and the corresponding murmur. The marked intensity of these signs tends to obscure the other lesions to which I have referred. They are well worth your study in this case.

In these cursory remarks my object has been to indicate to you the most important features presented by the various cases we have inspected, and to gather from them a few general lessons with regard to the origin, nature, combinations, symptoms and signs of cardiac disease. I cannot lay too much stress on the need for studying every case of this nature individually and specially. It is unwise to generalize regarding heart disease, whatever may be said for the process in other departments, and I particularly warn you against relying upon a mere theoretical knowledge of the subject, or upon statistics. It is only an intelligent comprehension of all the conditions actually present, founded upon adequate and systematic personal investigation, that will enable you to give anything approaching a reliable or confident prognosis in any particular cardiac case, or to venture upon its treatment with any reasonable hope of success, and indeed you will probably not uncommonly do your patients much harm if you act otherwise.

Moscucci treated two cases of malarial fever by means of *anagen* in daily doses of $15\frac{1}{2}$ to 31 grains, administered a few hours before the attacks. The splenomegalia present was cured in a month by means of an *ether* spray applied over the spleen, the tumour rapidly diminishing in size and the pain immediately disappearing.

(*Gaz. deg. Osp.*)

CASES DEMONSTRATED AT THE CLINICAL MUSEUM,

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Reported by J. T. CONNER, M.D.

Psoriasis Rupoides of the Senile Form.

A MAN, aged 70, sent by Dr. Barratt, had been the subject of this disease for four months. On the elbows the patches were of the ordinary kind, covered with silvery scales; but on the forearm they showed crusts like rupia. The palms were attacked, and the finger ends severely. The nails were black and much thickened. They had fallen off, from time to time, each in a single mass. There were patches on the ears, that on the left showed fissures. The knees and rest of the body were exempt. There was no history of syphilis.

Remarks by Mr. Hutchinson. The involvement of the palms, and the presence of rupia-like crusts, might suggest the diagnosis of syphilis; but the patches on the elbows are characteristic of non-specific psoriasis, and the fissures on the ear suggest the senile form.

Multiple Sarcomata of the Subcutaneous Tissue.

The patient was a woman, aged 43, sent by Dr. Stocker. Distributed irregularly over the trunk were a number of very hard nodules, varying in size from a pea to a chestnut. The largest was on the sternum. They were beneath the skin, but some were becoming adherent. A number were situated at the margin of the right breast, but only one near the left. The disease was of six months duration. The first growth appeared over the right infraspinatus muscle, but after a time it diminished, and entirely disappeared a fortnight ago. The woman was nursing an infant eight months old.

Remarks by Mr. Hutchinson. The case is one of great rarity and interest. We have Dr. Stocker's evidence that one of these hard lumps has entirely disappeared and left no trace. That is an event which we do witness occasionally in what is known as "withering sarcoma." I cannot doubt that the disease is of a malignant

nature, and it seems probable that the starting point has been the mammary gland, for in the peripheral lobules of the right breast are several hard masses. There is now general infection but no implication of the lymphatic glands as in carcinoma.

Elephantiasis of one leg following an injury; Amputation; Elephantiasis of the other.

The subject was a lady, aged 36, who slipped between the platform and the carriage at a railway station in December, 1893. The right foot was crushed, and a condition of elephantiasis of the leg gradually set in. A year afterwards the leg was amputated. Œdema of the left leg commenced in April last. It is now much enlarged and in a condition of solid œdema, but the skin is smooth and shows no lymphatic enlargements. The disease never extended beyond the knee in either leg.

Remarks. Mr. Hutchinson was of opinion that the accident set up a chronic erysipelatoid inflammation, which gradually passed into elephantoid œdema, and that the inflammation had spread by the lymphatics, and crossed the pelvis, thus producing a similar condition in the other leg. He had published several such cases. But they differed from this, in that the disease involved the thighs, which were quite exempt here.

Erythema Multiforme.

A girl, aged 12, sent by Mr. Hitchens, has been subject to attacks of this disease for four years, in the spring and autumn. The duration varied from a week to two months. The eruption was usually general, but now it is confined to the face and backs of the hands. It is symmetrical, simply erythematous on the hands, but on the face it consists of a group of slightly elevated pink discs, about the size of peas, occupying the centre of each cheek. At the onset of the attacks she loses appetite, and has tingling sensations in the parts where the eruption is about to appear. There was a strong family history of rheumatism and rheumatic gout.

Remarks. Mr. Hutchinson regarded erythema multiforme as a catarrhal disease. By this he meant, that the same causes which produced

catarrhs, in people with susceptible mucous membranes, produced this eruption in those with susceptible skins. The analogy was well borne out in this case by the recurrence of the attacks a particular seasons of the year.

Chronic Ptyalism in an old woman. (Hydrostomia.)

The patient was a woman, aged 68, sent by Dr. Conner. For the last ten years she had suffered from almost constant flow of saliva. The flow was worse at night, when in bed, than at any other time, and considerably interfered with her sleep. She went to sleep at ten o'clock, but was awakened about one in the morning, by the filling of her mouth with saliva, which caused a feeling as if she would be choked. After one the flow was so much that she got no further sleep, or at most dozed for a short time. Her mouth at this time felt very hot. In the day the flow was much less, and caused her but little trouble. Some of the secretion of the previous night was produced. It was a slightly opalescent fluid, and did not appear to differ from ordinary saliva. Her general health was good, but she had lost all her teeth, and did not use artificial ones at the onset of the affection. There were no signs of enlargement of the salivary glands, nor anything abnormal in the condition of the mouth, except that it was very wet. She had not been taking any drugs, and had never had mercury administered, as far as could be ascertained.

Remarks by Mr. Hutchinson. This is a case of great rarity, rarer even than the opposite condition Xerostomia, or dry mouth, with which, however, it corresponds very closely in all other circumstances—age, sex, and absence of disturbance of the general health. Dr. Conner has found a case, recorded anonymously in the *Lancet* (p. 845, I. 1892), which is similar in every respect to that of our patient. An old lady aged 75 suffered from constant flow of saliva, "during sleep and when awake." She had lost all her teeth, and did not wear false ones. There was "no depreciation of the general health," and "no tenderness of the salivary glands." No satisfactory explanation of the causation of these cases can be put forward at present. They are probably of nervous origin, and their subjects are always women.

A case of Syphilis showing early Tertiary Lesions.

A man, aged 23, sent by Mr. Jonathan Hutchinson, Junior, contracted syphilis three years ago. He showed ulcers at the angles of the mouth, like those in the secondary stage. The tongue was fissured, "lumpy," and bald from loss of filiform papillæ. Over the sacrum was a large "horse-shoe" patch of syphilitic lupus, with an abrupt, elevated, infective edge. On the back of the neck was a similar, but smaller circinate patch. On the chest was another patch of syphilitic lupus in a somewhat peculiar stage. There was a group of scars above the ankle, the result of deep ulcers.

Remarks by Mr. Hutchinson. The use of the term syphilitic lupus is justified by clinical facts. The lesions result from the effects of the syphilitic poison on a person predisposed to tuberculosis; which can often be shown by the existence of undoubtedly tubercular lesions in the patient, or other members of the family. Like lupus vulgaris, it can be cured best by destroying the infective focus. Cases which have been treated without avail internally yield at once, often to a single application of the acid nitrate of mercury.

ABSTRACTS of BRITISH MEDICAL ASSOCIATION MEETING PAPERS.

The Formation of Animal, Vegetable, and Mineral Calculi: Showing a Uniformity in their Construction, Irrespective of Chemical Composition and Locality of Deposition.

By GEORGE HARLEY, M.D., F.R.S.

THE communication illustrates the law of a "Unity in Nature," so ably advocated by Darwin, by showing how Darwin's theory of continuity in the animal and vegetable kingdoms may be equally philosophically carried into the mineral world as well; seeing that the morbid calculi met with in man and other species of animals, as well as in plants, are constructed exactly like the sands, gravels, and stones found on the sea-shore, and in the bowels of mother earth, and that all of them have but three special modes of for-

mation: by molecular agglutination, sedimentary stratification, or crystallization. Every calculus is not, however, necessarily entirely formed by one of these modes alone, or even by a combination of two of them; for in some instances—especially when the calculus is large—all three modes may take part in its formation. And not only so, but at different stages of its growth it may change backwards and forwards from one mode of construction to another. Beginning as a crystalline or a sedimentary, it may pass once or twice from the one to the other, and then end either as a crystalline or sedimentary concretion, according to what chanced to be its mode of development at the moment its growth was interrupted, either by its extraction from, or the death of its host. And not only so, but a calculus that began by a process of molecular agglutination, after passing through a stage of sedimentary stratification, may finally terminate in a crystalline stage of development, thus exhibiting during its career all three modes of formation. Moreover, large calculi, no matter whether they be urinary, intestinal, biliary or salivary, seldom or never possess a uniform chemical composition. This arises from the fact that the constitution of all morbid concretions varies with the life processes of their hosts. Consequently the clinical history of a patient may be as truthfully learned—at least, in as far as it goes—by the examination of a section of many of the large urinary calculi, as the geological history of the earth can be determined by an examination of the strata of rocks forming its crust. A calculus, for example, which began as uric acid may change in the process of its growth to oxalate of lime, and terminate in triple phosphates; or one that began as oxalates may end as urates, and *vice versa*.

It is a common idea that all calculi possess extraneous nuclei. This is, however, an error; for even among intestinal concretions, which are by far the most generally nucleated, many specimens are to be encountered where not a vestige of a nucleus of any sort is discernible. Such is the case in the majority of those beginning as agglutinated ones—as most commonly do animal and vegetable hair balls. At the same time, it is intestinal calculi which furnish us with the most characteristic examples of nucleated concretions, from the fact that all kinds of indigestible swallowed substances—such as fragments of stone,

glass, ivory, brass, iron, etc.—are prone to act as nuclei to food-calculi; and food-calculi are the kind usually met with in the digestive organs.

Lastly, the great law of uniformity in the construction of calculi was pointed out to exist by Dr. George Harley placing urinary, biliary and intestinal stones, whole, fractured and in sections, from men, monkeys, oxen, sheep and horses, with silicious tabasher stones from the hollow stems of the bamboo, mineral gravel from hard wooded teak trees, and crystalline oxalate of lime sand from Egyptian cacti, side by side with pearls from mollusca, marcasite balls—so-called “thunderbolts”—from chalk cliffs, kidney iron stones, Derbyshire spars, and crystalline nodules of wavellite rock; thereby demonstrating that in many instances it is impossible, from the naked eye appearances alone, to distinguish them. So closely, indeed, do some cholesterine gall stones resemble pearls and pisso-lite stones, as well as crystalline spars and wavellite nodules, that it is utterly impossible for any one—even an expert—to tell which is which, until they are taken into the hand and the marked differences in their respective weights are discovered.

The communication might therefore be not inappropriately called a contribution to the doctrine of “Unity in Nature,” seeing that it proved that there exists a uniformity in the construction of animal, vegetable, and mineral concretions, irrespective of their chemical compositions, modes of formation, and localities of deposition.

REVIEWS.

Manual of Physiology, with practical exercises.

By Dr. G. N. STEWART. Baillière, Tindall and Cox.

Price 15s.

The author states that “in this book an attempt has been made to interweave formal exposition with practical work,” and this he seems to us to have done in a most satisfactory manner. For us, however, the chief charm of Dr. Stewart’s book lies in the fascinating manner of its style. All the more intricate parts of physiology, e.g. the secretion of urine, the problems of metabolism and nutrition, are discussed with such pleasantness of diction and clearness of exposition that we involuntarily go on

reading page after page long after the mere duties of criticism are fulfilled. Whether this delightful style is that best suited to examination requirements we have some misgivings, but we sincerely trust that students will take to this book for pleasure, and extract for themselves the dreary 1, 2, 3, of examination tables. It is such a book as we should put into the hands of literary students pursuing physiology for its own sake. To all who are not reading for examinations (it is too good for such) we most strongly recommend this book for pleasurable and profitable reading. Dr. Stewart cannot be too highly congratulated on its production; we want more of such books in other branches of our studies. Fagge and Watson are its compeers in medicine.

Leprosy. By Drs. HANSEN and LOOGT; translated by Dr. N. WALKER, pp. 148. Wright and Co.

Nett, 10s. 6d.

For those who are interested more particularly in the study of this fearful disease, a careful perusal of this monograph seems to us to be indispensable. The authors are well known as specialists in the affection, one of them has practically devoted over twenty years to its investigation, and they have here presented a most detailed account of its various forms with their microscopic anatomy, diagnosis, prognosis and treatment; four exceedingly interesting tables (three on the relations of tubercle to leprosy, or rather on the association of the two diseases, and one on the results of isolation in Norway) are given at the end of the letterpress, and thirteen full-page plates of naked eye and microscopic anatomy most beautifully executed. The translator must be congratulated on the almost entire absence of awkward foreign expressions.

Text-book of Forensic Medicine and Toxicology.

By A. P. LUFF. Two Vols. Longmans and Co.

Price 24s.

It seems to be a growing fashion, which we cannot too strongly deprecate, for examiners to publish works on those subjects in which they examine; except for this fashion we can find no reason or excuse for the publication of these two volumes. We have no fault to find with the actual material in them, but there seems to be very little

that is not said equally well in other text-books; with Taylor and Stevenson, Tidy, and Dixon-Mann as works of reference, and with Guy and Ferrier, Husband, Abercrombie as smaller books for examination purposes, we cannot honestly find any room for the present work. We must make an exception to this rule, however, in the case of the chapters dealing with some toxicological points. From Dr. Luff's official position as analyst to the Home Office we should expect the most recent details in the detection of poisons present in minute quantities only, and we are not disappointed; except in Dixon-Mann's work on Forensic Medicine, we know no place where the differences between ptomaines and vegetable alkaloids are better treated of. The illustrations, too, are very good. For a real work of reference (which is a position we note Dr. Luff claims for it) the work is by no means exhaustive enough, but it will no doubt be read largely by students for those examinations for which the author is an examiner, and we certainly think it a very useful work for the purpose. It is beautifully printed, and very free from inaccuracies.

Report of the Surgical and Ophthalmic Cases admitted to the London Temperance Hospital. By Dr. L. WILDE, Registrar to the Hospital.

It is difficult to review a report of a hospital such as the one before us. We presume the object of the authorities is to show that alcohol is unnecessary, and that better results are obtained without its use; if this be so, it is very unfortunate that medical cases are excluded from the report. Considering that surgical cases are usually healthy except for the local lesion, a total mortality of 2.6 does not strike us as anything very brilliant. We notice a case of general tuberculosis reported as relieved, presumably sent home to die. We do not think that the cause of the sad of total abstinence will be much promoted by such figures.

Elements of Medicine. By A. H. CARTER. Seventh Edition. Lewis.

Price 10s.

As a student Carter was always our favourite "first reader" in medicine, and that subsequent generations of students have been equally favourably impressed, is proved by the publication of seven editions within fifteen years. The author's

own preface gives, we think, a true and just estimate of the scope of the work: "The object has been to provide broad truthful sketches of the various types of disease, as distinguished from a mere epitome or compendium of exhaustive treatises." This object has been well fulfilled, and it is this which elevates the work far above ordinary cram books; it cannot be crammed, and must be fairly read. We certainly think the new edition will be as popular as the previous ones, and deservedly so.

THERAPEUTICAL NOTES.

Typhoid Fever.—J. S. Carpenter, of Pottsville, Pa., gives the details of five cases of typhoid fever from an epidemic of forty treated by him in which he used *guaiacol* as an antipyretic. A maximum dose of 35 drops was employed, the average satisfactory dose being from 15 to 20 drops. When one application fails to cause a reduction of temperature within two hours, a second one may be made; and, according to the author, a reduced dose will effect as much lowering of body-heat under such circumstances as is desirable. His best results were obtained when the drug was combined with cold water baths or spongings.

(*Therapeutic Gazette.*)

Green Diarrhoea of Infants.—As soon as the first symptoms appear, Baratier, of Jaugny, France, gives a dose or two of ordinary purgative tea, and, when it has acted, he suppresses all food or drink, and gives regularly every two hours six tablespoonfuls of the following: *Fat bouillon*, 32 ounces; *glycerin* 3¼ ounces. The bouillon is made fresh every day, with 2½ pounds of beef-bones (no meat or vegetables) in 2 quarts of water, with a pinch of salt. It is boiled over a good fire for four hours until it is reduced to half the quantity, then allowed to cool, the fat skimmed off, and the *glycerin* added. This is essentially a *glycerophosphate* treatment within the reach of all. In the author's hands it causes the disappearance of the diarrhoea in a day or two; but it is continued three or four days, and then alternated with milk for about a week, when it is discontinued.

(*Trib. Méd.*)

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 13, 1895.

TWO LECTURES

ON

CARIES OF THE SPINE.

Delivered at the National Hospital for the Paralyzed and Epileptic, Queen Square, London, July 3rd, 1895.

By W. R. GOWERS, M.D., F.R.S.,

Physician to the Hospital, and Consulting Physician to University College Hospital.

LECTURE II.

GENTLEMEN,—I have, to-day, to complete what I desire to say regarding the subject of caries of the spine.

One or two other points regarding diagnosis deserve emphasis, and in connection with the recognition of this disease, as with the recognition of every disease, and especially of every organic disease, it is essential for you to acquire the habit of forming a mental picture of what is, or what may be the pathological state. This is important alike for diagnosis, for prognosis, and for treatment. The habit is of the utmost practical value. Its acquisition will save you from many errors, from many an error in diagnosis, many an error in your forecast, many an error in your treatment. Remember, therefore, what I said of the various ways in which caries of the spine causes paraplegia; that the deformity, the dislocation of the bones from their proper relation, may itself involve pressure on the cord; that the inflammation may spread through the membranes and involve the cord at that level as a subacute, transverse myelitis; that the swelling of the bodies, or a collection of pus or other like material may compress the cord, and, the bodies giving way and the collection of purulent material finding a new position, the cord may be released from pressure, so that, with the increase of the signs of the bone disease, may come a decrease or even disappearance of the palsy. Lastly, I mentioned that the most frequent mechanism of all is the accumulation of firmer inflammatory material, caseous matter outside the dura mater, which, slowly increasing,

presses on the cord. It follows from these mechanisms that the conditions of the spine, when paraplegia develops, are various, and among the facts which proceed from that, the most important, perhaps, is that the cord may suffer compression, and paraplegia may result, before any sign of caries can be detected on examination of the spine. It is true that the cases are rare in which there is no local tenderness, but they occur. There may be cord symptoms, seldom great, but still definite, without even tenderness to be elicited by pressure upon the spine. But that which is not present at one time may be at another. The great lesson for us from the fact I have mentioned is the need for repeated examination of the spine. It occasionally happens that the practitioner examines the spinal bones and finds no sign of disease, and dismisses caries from his mind. In two months time he takes the patient to a physician or surgeon, and, to his intense surprise, a fresh examination reveals obtrusive indications of bone disease. In any doubtful case the spine should be examined carefully at least once a fortnight until the nature of the case is otherwise clear. In these obscure cases, remember the great diagnostic importance of the radiating pain around the trunk at the level of the disease, due to irritation of the nerve roots. It may be on both sides, it may be on one only. Persistent pain around one side of the trunk at a definite level is seldom due to any other cause than organic irritation of the nerve roots. Neuralgic pains of the trunk are fugitive, inconstant, varying in time, and often in place; the pains of organic disease are persistent in the same spot. They do not always seem to be superficial in character, and hence they are sometimes mistaken for disease of the viscera. They may be ascribed to gastric or hepatic disease, and in one case the pain of caries was for long regarded as due to renal colic.

A great diagnostic difficulty is presented by the frequency of slight normal deviation from the common regularity of the spines. The weight that is to be attached to such deviation must depend on its correspondence with other symptoms. If you find that such pain as I have described is in the

course of the nerves which arise where there is slight bone irregularity, the latter deserves much weight. But the most significant coincidence is the relation of such pain to definite tenderness. Irregularity from caries does not occur without tenderness, but the latter is often present without irregularity. Correspondence in seat of slight symptoms is the most important of all the diagnostic elements in these obscure cases.

Remember, moreover, that tenderness, as a solitary spinal symptom, is of most significance when it is confined to one spot. In the cases of tenderness of the spine, which are due to other causes than organic disease—to neuralgia, to slight rheumatic affections, and to so-called "spinal irritation"—it is common to have more than one spot of tenderness, and multiplicity lessens its significance. Still more does variability diminish the importance of local tenderness. The direct effects of organic disease are fixed and not mutable.

Regarding the prognosis of the affection, remember that it is better the younger the patient; it is always grave in middle and late middle life. Never forget that caries occurs between 40 and 60 years of age, just as phthisis does. Many observers are thrown off their guard, and sometimes grave mistakes are made, in consequence of not remembering (occasionally from not knowing) that a patient 50 years of age may have simple caries of the spine, precisely such as occur in young persons. In the young, so far as paraplegia is concerned, the probability is in favour of recovery if proper treatment can be secured. But remember, also, that the danger of relapse at a future time is very great. I have had a patient in the hospital three times with complete paraplegia from caries, each time recovering.

Remember, further, that no condition of the bones that we are able to discern from the physical signs has much influence on the prognosis. I can, however, express no opinion upon the significance of the purely surgical element of the occurrence of abscesses; such cases do not come under the notice of a physician. The fact indeed is noteworthy. I doubt whether it is due only to the need for surgical treatment. It may be that, when liquid pus finds its way to a place at which it manifests itself as an abscess, there is not the local accumulation of the firm purulent caseous material which so often compresses the cord.

Lastly, no degree of paraplegia is inconsistent with recovery. There may be absolute motor palsy; there may be, although it is very rare, sensory palsy; there may be extreme spastic paraplegia, and yet there may be perfect recovery.

Regarding treatment. Two things, and two things alone, seem to be of supreme importance, and they are those that you all know. One is rest, and the other is the food- tonic treatment of cod liver oil and iron, to which should be added, when possible, fresh air. But rest and food are incomparably the most important. Every movement of the spine seems to involve local irritation, and an increase in the morbid process. It interferes with the stillness essential for healing. Rest is the first essential. The capacity of the diseased bones to recover—to recover ossification of the tissue so as to unite firmly, solidly, the damaged, destroyed, broken down remains of the vertebral bodies—is testified by every hump-backed man whom you see in the streets. Every one of those has had this breaking down of the bodies, the removal of their remains when they had undergone fatty degeneration, and had become discrete particles instead of a consistent mass. In each the process has had, as its sequel, a new formation of tissue, in which bone has been developed in abnormal position, has blended with that which has escaped, and has thus constituted a firm mass in the misshapen spine. To this process all displacement of the new tissue and new bone is prejudicial; rest is absolutely necessary. The patient's spine must be preserved for months from changes in the relative position of the structures. This can only be secured by horizontal rest, and, in disease of the cervical region, by an added apparatus for fixing the neck.

The question of the value of extension of the spine is one on which it is not easy to express a decided opinion. There is no doubt that some cases of caries of the spine, when suspended and extended, suddenly become able to move the legs, although they could not move them before. In such cases there must have been such displacement of the bones as to involve compression of the cord, and such displacement as extension could remove. Then, persistent rest has enabled recovery to occur without return of the compression. But it is very rare for this immediate effect to be produced. Still, I think

that in cases in which there is reason to believe that displacement of the bones has occurred, such as might be removed and rectified by suspension—a single experimental suspension is wise. I doubt the use of its repetition. I doubt also the wisdom of combining extension with rest. These damaged bones need to come together; the solid tissues which remain have to come into approximation, so that fresh solidity shall result with a minimum of new bony tissue. If we separate, by extension, the solid portions which remain, we increase the amount of new tissue which is needed to effect solidity. Encasement is, I think, useless during the stage of rest. It is of the utmost value when there is reason to believe that union has occurred, and the patient may be again permitted to resume the erect posture.

Of other drugs besides the food- tonic just mentioned, I can say little. I had once a case which filled me with hope. You may know that some years ago we heard a great deal of the value of sulphide of calcium in tubercular glands, and I gave sulphide of calcium to a patient with caries of the spine and paraplegia, who had been at rest for some time, and was stationary. Within a week of commencing the medicine, improvement occurred, and went on with the utmost rapidity. But, alas, in spite of many another trial, the case remains unique in my experience, and I am compelled reluctantly to regard it as an instance of the *post* and not of the *propter*.

One other measure, however, is certainly sometimes of service, and that is counter-irritation. In this disease, as in all others in which energetic counter-irritation is needed, for the spinal cord or for the bones, the actual cautery is incomparably the most useful. A hot point, a Pacquelin's cautery, or even a good sized copper wire, bent so as to form a small angle, may be made red-hot, and then drawn down three or four inches on each side of the spine; or it may be pressed against the skin quickly in spots about half an inch apart. It causes little pain—not enough to need an anæsthetic—and the local influence on the nerves should not be lessened by cocaine, because, as far as we know, it is through the nerve influence that counter-irritation does good.

Few and simple are the suggestions for the treatment of this formidable disease. It is a disease, however, in which much may be done,

only the "much" relates to one dimension. Length, not width, is the essential feature of the therapeutic measures. Such can tax the patience alike of those who try to give the necessary help, and of those who need it, and tax also the resources of the institutions in which alone the poor can gain a footing on the road to health. It is in this that we can often see most clearly the justification for the existence of the special hospital. The needful aid could be furnished in the general hospital, but on this there is a constant pressure of acute disease. To decide between the one and the other is beyond the capacity of the wisest; still more is it beyond the power of those with whom the decision now practically rests. Each life is equally precious, in our present code; to each, the health needful to use life is essential. It is well that, in some hospitals, the life that needs long treatment should not be sacrificed to secure a less certain good to another.

SOME SURGICAL CASES.

By STEPHEN PAGET, M.A., F.R.C.S.,

Surgeon to the West London Hospital and the Metropolitan Hospital.*

GENTLEMEN,—Let me first speak of two cases of fracture of the skull that came under my care last week. A boy, aged 11, was struck on the right side of the head with a brick thrown at him, and was admitted here at once. There were no symptoms such as we usually associate with severe injury of the head: no pain, no headache, no sickness, no confusion of thought: he sat up in bed, talked naturally, and hardly seemed hurt. There was extensive bruising of the right parietal region, and a small scalp wound, through which one could just feel a cracked edge of bone. But it was of course possible that there might be a depressed fracture concealed beneath the bruise: and an exploratory incision would at all events do the boy no harm. On reflecting the scalp, we

* West London Hospital Post-Graduate Course, October 9th, 1895.

found an extensive, deeply-depressed, irregular fracture of the right parietal bone, with the fragments so interlocked that it was impossible to raise them; and even after the trephine had been applied to the edge of bone just outside the fractured area, it was still necessary to cut away the locked edges of the fragments before they could be removed. The pressure on the brain was so great that there was no pulsation till all the fragments had been removed or levered up into place.

The boy has done very well. The wound healed quickly, he had no rise of temperature, and the opening in the skull is already smaller than it was at the operation ten days ago. The fragments of bone were not replaced; there is always the possibility that fragments thus replaced may fail to unite; but the boy will in time get a scar almost or quite as firm and safe as he would have had if the fragments had been replaced.

In strong contrast with this case, was that of a man admitted here last week under me. He was nearly 70 years old, and was drunk at the time of the accident. He was knocked down by a bicyclist. On admission he could stand with support, and tried to give his name and address; but when I saw him, some hours later, he was unconscious, with stertorous breathing, and a slow, full pulse. On account of these signs of compression, I at once prepared the patient for operation. But there were three points in his case which seemed to be against immediate operation: his pupils were small, equal, reacting well to light; there was some loss of power in the arm on the *same* side as the injury—(bruising of left side of head, loss of power in left arm)—thus pointing to injury of the right cerebral hemisphere by contrecoup; and those who had seen him at frequent intervals since his admission thought that he was less deeply unconscious than he had been a few hours ago. It seemed almost hopeless, in a man of 70, who was given to drink, to expect recovery after trephining on both sides; and on consultation I took the suggestion of two of my colleagues that it would be best to watch the case for a time. The next day he was in just the same condition; the day after he was weaker, and on the third day he died, without having recovered consciousness.

On examination, we found a very extensive fracture of the left side of the skull, passing upward

beyond the middle line of the vault, and downward along the groove for the left middle meningeal artery, across the base of the skull. The artery had been torn, and there was some hæmorrhage on this side, but not very extensive, between the skull and the dura. But on the opposite side, beneath the dura, there was a very extensive hæmorrhage. There was commencing meningitis over the upper cerebral convolutions on the left side. There was no laceration of the brain. The case was thus one of fractured base, with double hæmorrhage, ending on the third day in meningitis. Looking at the patient's age, and at the extent and severity of the injuries, it is evident that the case was practically hopeless; but I do not deny that an immediate double trephining, with incision of the dura on the right side of the head, would have been in accordance with the rules of surgery, and might perhaps have done good for a time.

The next case is a patient under the care of Mr. Eccles, who for eight years had suffered from a large ulcer at the back of the right leg, just below the popliteal space. The disease covered an area of 3 or $3\frac{1}{2}$ inches diameter; it had resisted various methods of treatment, and was evidently tuberculous. The whole ulcer was freely excised, and the wound was covered with large grafts taken by Thiersch's method from the front of the thigh. The grafts took well, and the whole surface is now healed.

The drawback to Thiersch's grafts is that the surface denuded by taking the grafts is in some cases exceedingly painful for several days; as painful as a burn or scald of the same size, and requiring the same careful dressing that a burn or scald requires. Mr. Wallis has invented a broad thin knife for the operation, with which it is possible to take grafts so thin that this pain is not inflicted; the blade has no thick back, so that the grafts may be readily slipped from any part of it on to the surface to be grafted. But if an ordinary broad razor, or an amputating knife, be used, the grafts may be picked up on a small round sponge, and thus transferred to the raw surface. And it is a good thing to cover them with Lister's protective, which allows all discharge from the raw surface to escape into the dressings without loosening the grafts. It is a mistake to cut one's grafts too big; one is sure, in attempting this, to go too deep, and to leave a very painful surface.

The next case is a child, two years old, suffering from that acute diffuse swelling of the cervical glands, which practically occurs only in infants and very young children. The infiltration of the surrounding parts is so extreme that the outlines of the side of the neck are lost, and there is a heavy ill-defined mass, extending from the angle of the jaw almost to the clavicle, like a rapid growth of sarcoma. The general health suffers badly; the children are pale, weak, and feverish; and I have known one case, in a baby, which ended fatally in a few days. The glands usually suppurate; and it is most important to let out the pus without delay, or even, in urgent cases, to make many small incisions in the hope of finding it. The reasons for this acute diffuse septic inflammation in and around the cervical glands in very young children are not clearly known; it can hardly be due to teething, for it occurs in babies; but it is probably secondary to infection from the mouth or nose; at least, nothing quite like it takes place in the axillary or inguinal glands of very young children.

In contrast to this acute inflammation of the glands in young children was the case of a man from whom I lately removed the inguinal glands on both sides on account of tuberculous disease. In one respect the removal of tuberculous inguinal glands differs from the removal of similar glands in the neck. The inguinal glands are best removed by making a free incision along the upper border of the swelling, not attempting to isolate the glands, but going down through the subcutaneous fat surrounding them, and at once exposing the front layer of the abdominal wall. When this is done, it is easy at once to peel off the whole chain of glands from the front of the abdominal wall and from Poupart's ligament. Below this level, the glands overlying the femoral vessels must be carefully raised, and there is often a gland lying just in the saphenous opening rather deeper than the rest. The sinuses that attend these tuberculous inguinal glands are often very extensive, and it is easy to overlook one of them, which runs downward and inward between the genital organs and the inner aspect of the thigh. Every sinus must be carefully laid open to the very end, and any incomplete treatment of these sinuses in the groin is sure to fail to cure them.

There is another condition of the lymphatic glands where excision is necessary, and I may,

perhaps, be allowed to quote my own case. So far back as May 22nd, I inoculated my finger over a case of suppurating hydatid of the liver. The inflammation of the finger was painless and indolent, and it was not incised till June 16th, when a little thick matter was let out. By this time the axillary glands were greatly enlarged, and by the end of the month they were a mass filling the whole axilla, but indolent, painless, and showing no sign of suppuration. It was thought that they might subside in fresh air, but during the whole of July they failed to improve; there was also fulness beneath the clavicle, plugging of a lymphatic vessel up the inner aspect of the arm, profuse perspirations, and a hectic temperature, often 103° at night; in short, it was a case of chronic septicæmia. At the end of July, Mr. Ballance, I am thankful to say, removed the whole mass of glands; some showed commencing central suppuration, others had not yet suppurated. With him for surgeon, a good recovery was almost a matter of course. In such cases as these, it is surely advisable to remove the glands, rather than to wait long for suppuration. The same operation has been done in acute septicæmia from post-mortem wound of the finger, but in these cases there is not much hope of recovery.

As regards the usual operation for tuberculous glands in the cervical region, there is one practical point worth noting. They are often enclosed in a well-marked thickened sheath, or capsule, separating them from the deep structures. If the surgeon does not very carefully define this sheath over the glands, and lay it open freely, he will fail to enucleate the gland quickly, and, keeping outside its sheath, will find that he is injuring the deep cervical fascia and needlessly exposing the deep vessels; but if he keeps just inside the sheath he will be able to shell out the glands easily and without risk of bleeding.

The following cases were also shown:

1. Congenital Displacement of the Femora. Mr. Eccles.
2. Large Chronic Abscess. Mr. Paget.
3. Congenital Nævo-Lipoma or Neuroma of the Forearm. Mr. Eccles.
4. Lymphangioma of the Knee. Mr. Bidwell.
5. Fractured Patella after operation. Mr. Paget.
6. Operation for Salivary Fistula. Mr. Bidwell.

A CLINICAL LECTURE

ON

EXOPHTHALMIC GOITRE,

By ARTHUR J. HALL, B.A., M.B. (Cantab), M.R.C.P.

Hon. Physician Sheffield Royal Hospital; Lecturer on
Physiology, Sheffield School of Medicine.Delivered at the Sheffield School of Medicine, on October
28th, 1895.

GENTLEMEN,—The case which I shall make the text of my remarks to-day is one that has been under my observation for a long time, upon which I have tried a considerable number of remedial measures, and which, unfortunately, still presents all the symptoms of what we know in this country as Graves' disease, and what is known on the Continent as the disease of Basedow. The history of the case is somewhat as follows:—

The patient, a single woman of 23 years of age, says that her neck has been swollen ever since she was twelve, but that it gave her no trouble until 1891, when she felt uncomfortable if her things were fastened at all tightly at the neck. In 1892 her friends noticed that her eyes were getting prominent, and she herself complained of difficulty in swallowing. In October, 1892, six months before she was admitted here under my care (March, 1893), she began to suffer from severe palpitation. She is not aware of any of her family having ever been "nervous," or having suffered from any form of goitre. On seeing her, the merest tyro in clinical work could at once make a diagnosis from the very evident cardinal signs, viz., exophthalmos, or prominent eyeballs, goitre, or enlarged thyroid gland, and tachycardia, or frequent heart-beat.

But although these three signs of Graves' disease are the most striking at first sight, yet on more close acquaintance with the disease, you will find that they by no means represent the whole of it, and the more you see of clinical work the more you will be convinced that Graves' disease is more common than text-books would lead you to suppose; but that a large number of the cases present, perhaps, one of the three cardinal signs in a marked

degree; another, only on careful examination; and the third not at all; whilst the other signs and symptoms, of which I shall speak shortly, are sufficient to stamp the character of the disease.

The order of the appearance of these three primary signs, and their relative intensity, varies very considerably; sometimes palpitation (which, by the way, is the subjective symptom felt by the patient, which, in this, though by no means in all other diseases, is usually coincident with increased frequency of heart-beat) is the first to appear and usually the most distressing; sometimes, as in this case, the goitre is the first; whilst sometimes attention is first drawn to the commencing illness by the friends noticing a change in the patient's face, owing to the prominence of the eyes. Moreover, these signs may alter very considerably in intensity more than once during the continuance of the disease, so that whilst the patient does not quite recover from them all, yet at one time the palpitation is less troublesome, at another the goitre is less enlarged, at another the eyes are less prominent. These transitory changes may coincide in time with medical or surgical treatment, and be ascribed to it, but they certainly also occur when no recognized treatment is being carried out, so that we must be guarded in distinguishing the *propter hoc* from the *post hoc*. We must also remember that even when the three signs are well marked they may all disappear for a time, so that the patient is temporarily better, and yet may appear again as well marked as ever.

What other signs and symptoms of Graves' disease are there besides these three? In a series of articles which appeared in the *Progrès Medical*, for December, 1894, M. Joffroy gives a list of about twenty of them, and although I have not seen them all in my cases, still there are a certain number which are fairly constant. The first of these I should place under the head of functional muscular disorders, not thereby meaning that the muscles themselves are diseased, but that some portion of the neuro-muscular apparatus is impaired. This head includes—(1) Tremors, sometimes limited to the arms, but often widely distributed over the voluntary muscles of the whole body, so that the patient, under the least excitement, or even apparently when merely making an effort to stand still, can be felt to be shaking all

over. These tremors are very fine, and succeed one another very rapidly; they are increased especially when attention is drawn to such attempt. They disappear when the muscles are at rest. In the orbicularis palpebrarum, as you will see in this patient, they are often very marked, as also in the arms when they are held out straight.

(2) Impairment of muscular power. This is seen markedly in the muscles about the orbit; for instance, there is frequently, as in this case, inability to completely close the orbicularis palpebrarum, or at any rate to keep it tightly closed for any length of time. This loss of power in the orbicularis is sometimes known as Stellwag's sign. It is not at all due to the lids being unable to meet owing to the protuberance of the eyeball.

There is another sign connected with the movements of the eyes and lids, the explanation of which is uncertain. The condition was first described by Von Graef, and bears his name; it is not always present. The patient is told to follow with her eyes the finger of the observer as he moves it from above her line of vision downwards: in a normal eye the upper lid moves downwards synchronously with the eyeball, but in some cases of Graves' disease the upper lid seems to hesitate before following the eyeball in its downward movement. It shows a loss of co-ordination between the orbicular and ocular muscles which is not difficult to conceive of, seeing how delicately these muscles are balanced, and how greatly, even in slight protrusion of the eyeball, the ocular muscles must be disarranged. In this patient you see Von Graef's sign well marked. Another sign, due, I consider, to functional muscular disorder, is the thin, weak, uncertain voice which so frequently goes with this disease. Others are occasional failure of the legs whilst walking, and, occasionally, contractures, though I have never seen the latter. Paralysis of the facial muscles, tongue, jaw, palate, or limbs are mentioned by Joffroy, but they have not occurred in any cases under my observation. The next prominent symptom is difficult to describe, but readily appreciable at the bedside—that is, the altered mental condition. Sometimes it is the first thing noticed by the patient's friends; there is an absence of repose, an irritable condition of the nervous system, a continual "nervousness," which often renders

the patient's life miserable, and entirely prevents application to anything. The patient feels hot and "throbbing," is often unable to sleep properly, and readily breaks out into a sweat.

Other functional disturbances are connected with the generative and digestive organs, amenorrhoea, vomiting, diarrhoea, etc.

On examining the patient you will usually find the thyroid unequally enlarged on the two sides, and feel a curious thrill over it, very marked in this case, and not felt equally well over the whole lobe. This thrill is of vascular origin, and is extra-thyroid. On feeling the pulse you will find often a pulse rate of 140 to 160, and yet dropping down to 100 or less at other times in the day; in this patient it is frequently over 160 when in bed. The skin is moist, often flushed, the cardiac impulse thumping, and sometimes a murmur is audible at the apex and over the front of the heart, although there is no valvular lesion.

The rest of my lecture, which is by no means long enough to adequately deal with such an interesting affection, must be devoted to telling you very briefly and concisely what are the views which at present excite much discussion and controversy as to the nature and ætiology of Graves' disease. The first difficulty arises from our inability at present to classify it. The chief number of the symptoms and signs point to a nervous origin, as you will already have noticed; thus, the altered mental condition, which sometimes develops into lunacy, the palpitation, tremors, loss of power, paralysis, all belong to the category of nervous symptoms.

But no disorder of the central nervous system that we know of produces enlargement of a gland as a constant and sometimes first sign; that is the difficulty for the nervous origin supporters to get over. There are some, however, who hold that the disease is primarily one of the thyroid gland, of the nature of an excessive functioning, or hyperthyroidation. They base their theory on the following: (1) The thyroid body is always enlarged in Graves' disease—though sometimes this enlargement is so slight as to be hardly perceptible; (2) it is not merely increased in vascularity, it is altered in structure so as to be hardly recognizable under the microscope as thyroid gland; (3) the remote effect on the nervous system of hyperthyroidation is a train of symptoms such as are

found in Graves' disease, tachycardia, restlessness, muscular tremors, etc.; (4) full doses of thyroid extract given for other purposes produce also tachycardia, restlessness, and other nervous symptoms.

Now about these two theories the time is not yet ripe for any dogmatic statement. There are many facts recorded which it is useless to bring before you here, though some are of extreme interest. I can, however, say this, that in three of my cases where I have had an opportunity of examining the thyroid gland microscopically, I have been struck with the comparatively slight increase of vascularity in the substance of the gland, and the great overgrowth and alteration in character of the gland tissue. Also in one case of psoriasis, in which I gave somewhat large doses of thyroid extract, symptoms of melancholia set in, and in many others palpitation, throbbing all over, and nervousness have been produced. Further than that I shall not at present commit myself, nor is it necessary for you to go at this stage of your studentship.

I must now speak to you of the treatment to adopt. Leaving to your text-books the preliminary generalities of hygienic methods, let us consider here only particular kinds of treatment: (1) Nervine sedatives: amongst many, potassium bromide and belladonna are the chief I have tried. The former has signally failed, the latter occasionally been beneficial, and I always begin with that, and give it a thorough trial. (2) Local application of cold by Leiter's tubes to the præcordium; this in some cases has caused much relief, but it is only temporary. (3) Faradism to the pneumo-gastrics—one pole on the neck, the other over the heart. This again has caused considerable temporary improvement. (4) Removal of a portion of the thyroid gland. This operation has been twice performed on this patient at my request by my colleague, Dr. Sinclair White, and as the question of its success or failure involves so important an issue, I must say a few words as to the result. That the disease has not been removed, or even permanently ameliorated by the removal of the right lobe and isthmus of the thyroid is painfully evidenced by the patient's present condition. But there are several circumstances connected with it which call for careful notice. The first operation was performed on March 1st, 1894, and two weeks later I have a note, that "I never

saw her so quiet, or the eyes so little prominent since she has been under my care; she feels much better, and her palpitation is very much less." Unfortunately she gradually went back, the old troubles returned, and thinking that perhaps the operation had not been sufficiently radical, I asked Dr. White to remove the right lobe, which he did very successfully on April 6th, 1894, and up to April 24th, when she went out, she was distinctly better in many respects, though less so than I had anticipated from the result of the previous operation. By the middle of May, however, she was as bad as ever again, and has remained so ever since. During the last year the left thyroid lobe has undoubtedly increased considerably in size, and the delicate question arises, are we justified in removing the greater part of this left lobe, or even the whole of it? She herself wishes it, but we have to carefully consider the danger of setting up myxœdema if we remove the whole. Could we keep this in check by giving her thyroid extract? Probably we could. But shall we relieve her of her present distressing malady by removing it? These are questions to be weighed carefully before deciding. For my part I should put it before the patient and her friends in this way, "If the operation is performed, you may be completely cured of your present affection, or you may be as bad as you are now, but there is no reason to suppose you will be worse. You will run the risks attending all operations, and you may afterwards develop other symptoms which will necessitate your constantly taking preventive measures, in themselves very simple." For myself, I should give my consent, but, I think, only to complete removal; any further partial removal seems unwarrantable. How we may settle it finally I do not know, but I want to tell you, before closing, of a curious feature which was present in two cases of Graves' disease in which I made post-mortem examinations, and in at least two cases which have been recorded elsewhere—namely, not only the persistence of, but the unusually large size of the thymus gland—this surely cannot be merely a coincidence. But of its meaning I know nothing; I tried, in one or two cases, feeding on extract of thymus gland, but without the least benefit, and I merely mention the fact of its presence in these autopsies as a curious fact worthy of your remembrance.

CLINICAL REMARKS

ON

THE USE OF MYDRIATICS IN THE EXAMINATION OF EYE CASES.*

By SIMEON SNELL, F.R.C.S. Edin.,

Ophthalmic Surgeon to the Sheffield General Infirmary ;
Lecturer on Diseases of the Eye, Sheffield School of
Medicine, &c.

CERTAIN drugs when brought into contact with the surface of the eyeball are absorbed and occasion dilatation of the pupil. Most of them also cause paralysis of the ciliary muscle. These drugs are called mydriatics. They are largely employed in eye diseases, and before and after certain operations. It is my purpose now, however, to direct your attention to mydriatics as used in the examination of eye patients. They may be required for two reasons. Firstly for their dilating action on the pupil alone, and secondly in refraction cases for their effect on the ciliary muscle.

Let me first, however, mention briefly the mydriatics which are in vogue. Among the best known are atropine, homatropine, cocaine, duboisine, hyoscine, daturine, and scopalamine. Mydrine is a new mydriatic to which attention will again be directed ; up to the present it has been little used. Scopalamine has of late been employed a good deal. Daturine is obsolete, and the same may be said of duboisine, which was so apt to cause toxic symptoms, sometimes of a very alarming nature, that it has ceased to find much employment in ophthalmic surgery. Hyoscine, also, a very active and powerful drug, has for similar reasons been abandoned in practice. Cocaine differs from other mydriatics in that its action on the accommodation is practically nil. Moreover, the mydriasis it produces is unlike that occasioned by atropine and the others, for under the influence of a general anæsthetic it is observed to disappear, but as recovery from the anæsthetic takes place the pupil again becomes dilated. This fact was pointed out by me † just after the introduction of cocaine as a local anæsthetic in ophthalmic

surgery. I mention it here particularly as it is important not to rely on cocaine mydriasis in any operation in which a dilated pupil is necessary, say, for instance, needling of a soft cataract, and for which it has also been deemed advisable to give chloroform or ether. Cocaine, however, added to other mydriatics increases their effect on the pupil and on the ciliary muscle.

All these drugs may be employed in solution in distilled water, or with olive or castor oil, or vaseline as vehicles. It has been held that these latter media by retaining the drug longer in contact with the surface of the eyeball produce a more constant and better result. Another method is to use gelatine discs containing the drug.

We may now discuss the purposes for which mydriatics at present claim our attention.

1. In suspected cases of iritis the use of a mydriatic is often desirable to render evident the presence of adhesions of the pupil to the lens capsule, but this point has already been mentioned when considering with you iritis and its treatment. It will only be necessary, therefore, to say that atropine in the form of the sulphate is most generally employed for this purpose, and the strength required is four grs. to the ounce, though a solution half as strong if used several times in succession will often suffice for diagnostic purposes. Cocaine added to the atropine will increase the effect of the latter, a one per cent. solution will be strong enough. Scopalamine is at present vaunted a good deal for its efficacy in freeing iritic adhesions.

It is also often necessary for the complete examination of the interior of the eye, to have the pupil dilated, and thus it is frequently required for ophthalmoscopic examination and for ascertaining the condition of the crystalline lens and the vitreous humour. For these purposes it is desirable to employ a mydriatic which acts as little as possible on the ciliary muscle, and the effects of which pass off quickly. Atropine has the disadvantage, even in a weak solution, of affecting the accommodation, and besides this, its action is a long time in being recovered from. If, therefore, it is employed for the purposes just indicated, the solution should be a weak one, one gr. to the ounce, for instance, or, though much slower in acting, a solution as weak as one gr. to eight ounces of water will occasion sufficient dilatation of the pupil for ophthalmoscopic examination, and the

* Portion of a lecture delivered at the Sheffield General Infirmary, July 17th, 1895.

† "British Medical Journal," 1885, Vol. 2, p. 153.

effects will much more quickly pass away. Cocaine of a strength of 2 per cent. is useful, but the dilatation will remain for twenty-four hours or more. Either atropine or cocaine will dilate the pupil sufficiently in from twenty minutes to half an hour. Scopolamine is asserted to act like homatropine, but more powerfully on the ciliary muscle.

My own practice has been to employ either homatropine or a weak solution of atropine, and less frequently cocaine alone. A new mydriatic called Ephedrin is asserted to occasion mydriasis, which remains at its maximum for half an hour and then subsides, disappearing in two hours after the first use of the drug. Combined with one per cent. of homatropine the effect is increased, but the return to the normal state is a little delayed. The following formula gives the proportion in which it is used:—Ephedrin hydrochlor., 1'00; homatropine hydrochlor., 0'01; aq. dist., 10'00.* This combination is manufactured by Merck under the name of Mydrin. The ephedrin-homatropine solution does not influence the accommodation in the least, and though the mydriatic action is quite rapid and powerful, still the pupil never entirely fails to react to light. After a single application of this combined solution to the eye, the pupil begins to dilate, on an average, within eight minutes and a half (varying from six to thirteen minutes), and attains its maximum dilatation within half an hour, the average being thirty-four minutes, varying from twenty-three to forty minutes. Within an hour after the application the pupil slowly begins to contract, and after the lapse of from four to six hours, has again attained its normal size. The greatest dilatation continues for about half an hour, varying from fifteen to forty-five minutes, the average being twenty-nine minutes. At the maximum dilatation the pupillary diameter measures from five to six millimetres (average, 4.5 to 7 millimetres), which is sufficiently large for diagnostic purposes. My experience with mydrin is, up to the present, a favourable one.

2. In testing refraction it is necessary that the drug employed should exercise a temporary paralyzing effect upon the ciliary muscle, in order to reveal the actual refraction apart from any influence of the accommodation. Atropine answers

this purpose well. If a 4-gr. solution of the sulphate of atropia be instilled, the pupil will become dilated in from twenty minutes to half an hour, but it will take about two hours for the ciliary muscle to be affected. The minimum time, therefore, to allow for the action of atropine in a refraction case is two hours; but it is seldom that such a period is sufficient, and particularly is this the case with regard to children. It may be deemed as essential that the application of the atropine should, as a rule, be repeated several times. My custom with children is to prescribe the use of the drug for two days, to be instilled twice daily, the last instillation on the second day being more than two hours before the time fixed for the examination. In cases also where spasm of the ciliary muscle is present it will often be necessary to continue the use of the atropine for several days. The great disadvantage of atropine, however, is the slowness with which its effects pass off. Its action on the ciliary muscle will be somewhat lessened in about three days after the drug has been discontinued, but it will take a week, or more often ten days, for the eye to recover its normal condition. These drawbacks are so great that they have acted as a hindrance in many hands to the systematic use of a mydriatic for refraction cases. To the artisan the incapacity occasioned, even when the eyes were treated singly, is a matter of considerable moment; and to the well-to-do the lengthened period of inability for close work is disagreeable and much objected to. In my opinion the regular testing of refraction under the influence of a mydriatic in young people is of the utmost importance, and it is required more frequently in adults than is the general practice; a mydriatic, however, is desirable whose effects will pass off quickly, and for this reason homatropine, in my practice, has for long replaced atropine save in exceptional cases.

The action of homatropine differs from that of atropine in being more rapid and more evanescent. Under its influence the pupil becomes dilated in about twenty minutes, and the accommodation is paralyzed in an hour or a little more. The hydrobromate is the salt generally used, and the strength of the solution 2 to 4 per cent. It should be instilled three times at intervals of quarter of an hour, and before testing an hour and a quarter to an hour and a half must elapse after the first instillation of the

* George F. Suiker, M.D., "New York Medical Journal," June 8th, 1895.

drug. The addition of cocaine increases the efficacy of the homatropine. My own experience with the combination is most satisfactory, and for the majority of refraction cases its action on the ciliary muscle is sufficiently great. A better mode of using homatropine and cocaine is, however, in the form of gelatine discs (Casey Wood). The gelatine slowly dissolves, and allows the mydriatic to remain for a longer time in contact with the surface of the eyeball, and thus the action produced is more certain as well as more powerful than is obtained by using a watery solution. After considerable experience with this method I can speak confidently of its value. The gelatine discs to which I have referred are made by Wyeth Bros. according to the suggestion in the first instance of Dr. Casey Wood, of Chicago. Each disc contains 1.50 gr. of homatropine and the same amount of cocaine. The patient is directed to look upward, and then on drawing down the lower eyelid a disc is allowed to slip on to the ocular surface; the lid is next permitted to slide gently back, the disc remaining between it and the globe, and the patient is desired to keep the eyes closed for a short time. A second disc is inserted in a quarter of an hour, and the action of the ciliary muscle will be set on one side, and the eye ready for testing in seventy to ninety minutes, the usual time allowed by me being an hour and a quarter. In cases of suspected spasm of the ciliary muscle the two discs may be followed by inserting another containing 1.25 gr. of homatropine only, as suggested by Dr. Casey Wood. The effects of the homatropine used as just mentioned will last from twenty-four to thirty-six hours, and the period can, if it be thought desirable, be further abridged by inserting eserine. The rest insured to the ciliary muscle, is, however, of therapeutic value, and generally speaking should not be curtailed. Instances will be met with in which the combined action of the homatropine and cocaine is not sufficiently powerful, and a resort to atropine will be necessitated. The combination, however, suffices in the great majority of cases, and the prompt action and quick disappearance of its effects are features greatly inducing to the regular and systematic use of a mydriatic in refraction cases.

A caution is necessary as to the use of mydriatics. All of them have a tendency to increase

the tension of the eyeball, and hence in incipient glaucoma their employment may precipitate an outbreak. It is well, therefore, to use them with care to patients over 40; to ascertain previously the tension of the globe; to inquire as to the presence of prodromata of glaucoma, such as rainbow colours, &c.; to employ weak solutions when practicable, and to instil afterwards either eserine or pilocarpin (myotics). Atropine and those mydriatics whose action is prolonged, are perhaps more likely to be injurious than others like homatropine and cocaine, whose action is more transient. In practised hands these drugs may be used with advantage only, but caution as to their employment is desirable, as, by no means infrequently, cases of glaucoma are met with in which the affection has either been induced or aggravated by the injudicious use of atropine or one of its allies.

THERAPEUTICAL NOTES.

Facial Erysipelas.—H. de Brinon, of Moulins, has had excellent results from the treatment of erysipelas of the face by the *sublimated spray*. Instead of an *etheral solution* of 1 to 100, as recommended by Talamon, he uses a solution of 1 to 1000. The burning sensation complained of when the former is employed is not experienced, while the method remains equally efficacious.

(*Concours Méd.*)

Furuncle.—To prevent secondary attacks, so frequent in these cases, wash with a 1 to 1000 solution of *sublimated*; then—with *carbolic acid*, 1 part; *camphor*, 2 parts—wash the healthy skin in the neighbourhood of the primary furuncles, wipe the surface carefully with absorbent cotton, and paint with *traumaticin* or *steresol*. When this has been thoroughly applied treat the furuncle as may be indicated. Apply a new coat of the varnish from time to time, and warn the patient of the necessity of not rubbing the affected part, of keeping the nails clean, of frequently washing the hands in an antiseptic solution, etc., in order not to carry the staphylococcus to another part of the body; also sterilize clothing brought in contact with the affected part.

(*La France Méd.*)

Insomnia of Neurasthenia.—Claus considers *trional* as the best and least dangerous of the hypnotics in the insomnia of neurasthenia. It should be administered before retiring, in varying doses, according to the degree of insomnia and the age of the patient; ordinarily $23\frac{1}{4}$ grains is sufficient. Of thirty cases observed by him the results were uniformly favourable.

(*La Flandre Méd.*)

Malaria.—Verrier advocates the use of local douches in the treatment of intermittent fevers and malarial cachexia. He calls attention to the fact that a too powerful local douche, instead of being antihyperæmic, produces a congestive effect by the prompt and energetic reaction which follows it; while a too weak general douche produces congestion instead of revulsion. If an antipyretic or antiperiodic effect is desired, the douche should be a general one, very strong, lasting from fifteen to twenty seconds, and given a quarter of an hour before the expected febrile attack. In such a case the douche exercises a short and very strong perturbatory action on the nervous system, driving the blood from the periphery toward the centre; and this movement of concentration is immediately followed by an easy and strong reaction, causing the blood to flow towards the periphery. A single such douche, properly administered, is often sufficient to cut short a regular intermittent fever, no matter what its origin.

(*La France Médicale.*)

Malarial Hepatitis.—Robin, of Paris, finds that, while sulphate of quinine is often without effect in congestion of the liver of malarial origin, quinquina gives good results when associated, in a prolonged course of treatment, with arsenic, iron, and hydrotherapy. He lays down the following plan of treatment: 1. *Arsenate of sodium*, $\frac{7}{8}$ grain; *water*, $9\frac{1}{2}$ fluid ounces. A tablespoonful morning and evening. 2. Yellow and red *cinchona powder*, each 1 drachm. To make 30 powders, 1 powder to be taken daily in one or two doses, in black coffee, preferably after meals; or, if not well supported, 8 or 10 pills daily, each pill containing gray and yellow *cinchona powder*, each $1\frac{1}{8}$ grains; *extract of quinquina*, 4 grains. Make 60 such pills. 3. Every morning a cold douche,

commencing by a jet directed to the spine and ending by a powerful one on the liver and spleen. This treatment is continued for a month, when arsenic and quinquina are replaced by an iron preparation for a month, the former treatment being again resumed and alternated monthly until all symptoms have disappeared.

(*Bull. Gén. de Thé.*)

The Value of Alumnol in the Treatment of Gonorrhœa in Women.—Dr. Gommard employs (*La Presse Médicale*) alumnol in solution of one to five per cent. in vaginitis or endometritis of gonorrhœal origin, by injections, using the intra-uterine catheter of Bosman. The injections were made every three days, and after three or four injections the secretions had markedly decreased.

The following precautions are to be observed in the treatment:—

1. The solution should be fresh.
2. Dilate primarily the cervix uteri.
3. Irrigate under a feeble pressure, to avoid forcing the contents of the uterus into the tubes or peritoneal cavity.

A Substitute for Sulphur Waters.—It is pointed out in *La Médecine Moderne* for June 1895, that the natural sulphur waters do not keep well during transportation, and, as a result of this, are not efficacious, except at their source, chiefly through the fact that the sulphur compounds are altered. At the same time the advantages to be obtained from the use of sulphur water are so great that it is worth while to attempt to produce a substitute, and it is thought that a solution of hyposulphite of sodium dissolved in glycerin and water is therapeutically active. An advantage in having the glycerin present in the solution is that it remains unaltered from several months to a year.

The following formula may be employed:—

- Crystalline hyposulphite of sodium, 3iiss;
Distilled water, 3x;
Pure glycerin, 3v.

The hyposulphite of sodium is to be dissolved in hot water and the glycerin added. A teaspoonful of this solution mixed with a quart of water charged with carbonic acid gas forms instantly an artificial sulphur water.

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 20, 1895.

TWO CLINICAL LECTURES

ON

ENTERIC FEVER.

Delivered at the Middlesex Hospital, on October 26th, and
November 15th, 1895.

By **SIDNEY COUPLAND, M.D., F.R.C.P.,**

Physician to the Hospital.

LECTURE I.

ULCERATION OF LARGE INTESTINE IN ENTERIC FEVER—DIFFICULTIES IN DIAGNOSIS— EHRlich's TEST.

GENTLEMEN,—The subject of enteric fever is so wide, that it is practically impossible to give a sufficiently full account even of its clinical history in a single lecture. There are so many variations in its mode of onset and its course, and so much difficulty often in its early diagnosis, that it presents abundant clinical material for consideration, and one is always obtaining fresh experiences. A few of the cases may certainly run the typical course with which the text-books have made you familiar, but many, perhaps most, exhibit departures from this type.

I will preface my remarks to-day by showing you the intestines from a case recently in Hertford Ward, a case which exhibited some uncommon features. The lower part of the small intestine, and nearly the whole of the large intestine display a considerable amount of ulceration. The lesions in the small intestines are situated upon the free surface of the bowel, many of them obviously arising in Peyer's patches, and on closer inspection are seen to be ulcers of very irregular shape, not involving the whole patch; moreover they have sharply defined margins which are deeply undermined. The bases of the ulcers are free from any adherent sloughs, and in some of them the transverse muscular fibres of the bowel are distinctly seen; others seem to be covered over by some smooth, semi-translucent material. In the

small intestine, the ulceration is most marked, at the termination of the ileum, just above the ileo-cæcal valve, though it extends for some distance up the ileum. In addition, there is also distinct swelling of the solitary follicles. But the changes are most marked and most peculiar in the large intestine; here the mucous membrane is unduly thickened, more in some places than in others, and there are large irregular tracts of ulceration which give the bowel a worm-eaten appearance—a most extensive destruction of mucous membrane.

The general aspect of this colon reminds one more of dysentery than of enteric fever, and this is so even where the ulcers are more widely separated; there is the same punched-out appearance, and the edges are very deep. This specimen from the museum shows an equally extensive ulceration of the large intestine in typhoid fever. You are probably aware that involvement of the colon in the disease is comparatively rare, but my impression is that such extensive ulceration has of late been more frequent than it used to be. When I was engaged in making post-mortem examinations, in very many of the cases of typhoid fever the colon was intact, but of late years several of our fatal cases have shown extensive ulceration of the colon.* This is a feature which, so far as I know, has not been commented on, and it may be peculiar to our experience. I may remind you that the stages of ulceration in typhoid fever are very definite. I have brought some drawings to show these different changes in the ileum. Succeeding to congestion there are swelling and infiltration of Peyer's patches and solitary glands, which implies the formation of a large amount of new material. The cell proliferation occurs with such rapidity that it blocks the vascular supply of the patch, and soon falls into necrosis, which mostly begins in the central

* In the twenty years (1874 to 1893), in 117 cases of enteric fever examined after death at the Middlesex Hospital, 44 showed *some* ulceration in large intestine; in only 14 was this ulceration *widespread*—i.e. about 12·4 per cent. of all examined. In the first decade, of 73 examined such extensive ulceration in large intestine occurred in 5 or 6·8 per cent.; in the second decade, in 9 out of 44, or 20 per cent.

parts of the infiltration. Later, the sloughs due to this necrosis come away, leaving ulcers with undermined margins. These ulcers heal by granulation, and their cicatrization occurs without causing any contraction of the bowel whatever. It is of some importance to realize that the intestinal changes in an ordinary case of typhoid fever probably run this very definite course, although it is seldom that all the infected tract is simultaneously involved. In fact one might take a typical temperature chart of enteric fever and divide it up into periods which would fairly coincide with the successive changes which occur in the intestine. During the first week, when the temperature is rising, inflammation of Peyer's patches is extending. The next week—the period of continued fever—the inflammation is more intense and sloughing is commencing. During the third week the sloughs are separating, and now there is a period of remittent fever, which is often attributed to absorption of material from the ulcers. Then follows healing, so that taking the chart as our guide, we withhold solid food until after a few days' normal temperature we may hope that every lesion is healed, so as to avoid the risk of irritating unhealed ulcers.

But many cases of typhoid fever do not really run such a simple course, and we have to ask the reason they do not do so, and whether we can learn anything from the anomalies they present. Inspection of this intestine would lead us to say that the healing process in this case had only just commenced. If it were a normal case of typhoid fever, it would be the appearance presented in the third or fourth week. So far as we can ascertain, however, the patient succumbed after six weeks' illness. It is true that the man himself said he was only taken ill about five days before he came into the hospital; but his mother, who had evidently been more observant than the son, noticed that he was unwell fully a fortnight prior to coming for advice, and, bearing in mind the insidious character of the onset of enteric fever, I incline to regard the mother's version as correct.

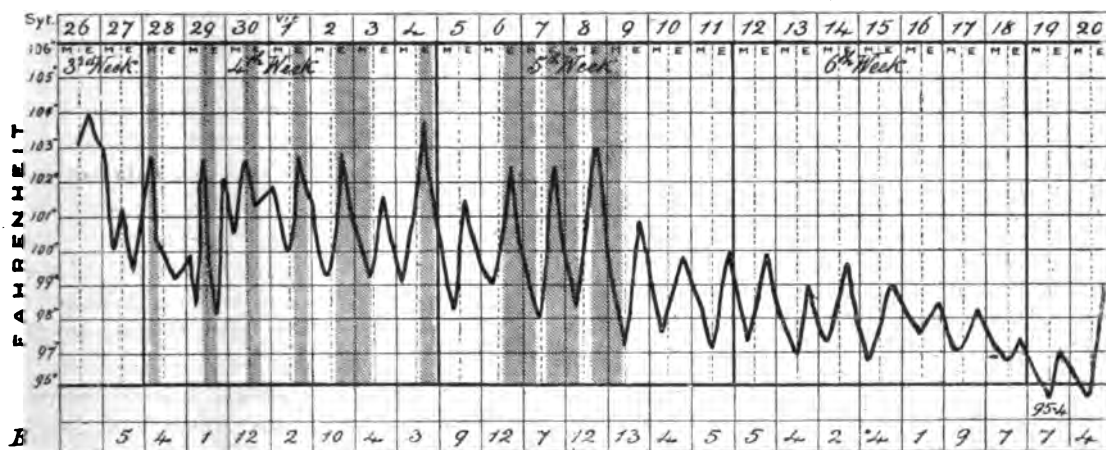
The man was thirty-five years of age, unmarried, and lived alone with his mother, whose only child he was. The whole of his life had been spent in London, and, except for occasional headache, he had suffered no illness of importance. Up till recently he had been of temperate habits, but the

receipt of a small fortune appears to have led him latterly to take to drinking rather heavily, almost exclusively malt liquor. He was admitted on September 26th, suffering from fever and diarrhoea, the latter having commenced abruptly on the 21st, and it was for this he came to be treated. Now, enteric fever very rarely begins abruptly with diarrhoea; ordinarily, as I have said, the onset is extremely insidious, with little or no disturbance of the intestines. The diarrhoea increased in severity, and could not be checked. Then we learnt that he had, at the end of August, been to Margate for a holiday. It is curious that another enteric patient, who was in the hospital at the same time, had also contracted his illness, apparently, at the same health resort. Besides diarrhoea, the patient under consideration had, during the preceding week, suffered from headache, and had a slight cough, but no epistaxis. He was a muscular, well-developed man, in a state of great prostration, lying on his back; his face was flushed, but his manner was quiet and rational. The pupils were contracted, and there was tremor of the lips, and of the tongue when protruded; his hand also trembled when he held it out, symptoms which pointed to very marked nervous and muscular prostration. The temperature was 103° , and the tongue dry and furred. He had told us that the diarrhoea had been increasing all the week, and had become almost uncontrollable; consequently we looked at the abdomen, and found considerable distension, and that it was tympanitic, but no gurgling could be made out, nor was the abdomen tender in any part. The margin of the spleen could just be felt below the ribs. As far as we could make out there was no obvious enlargement of the liver. We searched diligently for rose spots, and found an abundant crop on the surface of the abdomen. These spots seldom appear before the end of the first week; most commonly about the tenth day, and they will continue to come out in successive crops for variable periods. Some cases of typhoid fever go through their career without any visible eruption, but where the crop is abundant they may continue to appear until the third week or even later. The pulse was 124, regular, full, and soft; the first heart sound was short, indicating some myocardial enfeeblement. The urine was sp. gr. 1018, contained no albumen, and gave the reaction associated with the name

Ehrlich. He was put on opiates, to check the diarrhoea, if possible, and as his temperature rose to 104° he was sponged, with but slight effect. Towards evening he became very delirious; he had delusions, in which he got out of bed, and was very noisy. In view of the history, one might think this could be explained by alcoholism, but it was much more probably the result of his fever. He was, of course, isolated, and then became quieter, especially after he had had some morphia. As the fever remained at 103° he was "ice-cradled," i.e. surrounded by a cradle in which the air is cooled down by the suspension in the cradle of trays filled with ice and salt; and this

tents. As the opium given by the mouth alone did not check the diarrhoea, a starch and opium enema was administered. This brings us to October 6th. His mental condition remained abnormal for some time; he had delusions occasionally, but there was no recurrence of the delirium. From the end of October onwards, as you will see by the chart, the pyrexia rapidly abated; not only did the temperature reach normal, but, more often than not, it was subnormal; and with this the man seemed to waste even more rapidly than when he was feverish. The diarrhoea was still bad, but varied very much; at this period it was never more than

TEMPERATURE CHART OF CASE OF ENTERIC FEVER WITH ULCERATION OF COLON.



THE SHADED PORTIONS INDICATE PERIODS OF "ICE CRADLING."

undoubtedly had effect in controlling his fever (see temperature chart). The cradling was discontinued whenever the fever subsided to any extent. A glance at the chart, which I hand round, will show the result of the ice-cradling, and it shows that the antipyretic effect of cold air is much more effectual than one might be inclined to think. However, it was not so much the intensity of his fever that we had to deal with, as the marked nervous prostration, and the obstinacy of the diarrhoea. This latter subsided for a few days, then increased again. About September 30th the motions became very offensive, whereupon we gave him 5 grs. of β -naphthol, a drug which markedly controls the foetor of the intestinal con-

seven or eight motions a day, whereas earlier in the attack ten or twelve times a day was frequent. The abdomen became extremely *retracted*, more like what is seen in the last stage of tubercular peritonitis. Further, his face and hands became very cyanosed, but his pulse remained of fair strength and volume, and did not seem to correspond to the apparent collapse. He was drowsy and quiet, and his tongue was dry, whilst stimulation altered his condition but slightly. This state commenced about October 14th, and he died on October 20th, his temperature having risen to 99° . The condition of the intestines shows how almost inevitable this termination was. The very extensive ulcerative process in the large intestine

sufficiently accounts for the extreme obstinacy of the diarrhoea, and for the fact that it persisted so late in the disease. But yet that does not seem to me to alone explain the fatal termination. You know that usually the direct causes of death in typhoid fever—those connected with the intestines—are perforation or hæmorrhage, which occur when the sloughs are separating. In this case the ulceration was very deep, but, fortunately, during life there was no perforation, though this must have been on the point of occurring more than once. The ulcers were retarded in their healing, and their presence perpetuated the diarrhoea, and the extreme prostration and collapse must be ascribed to this as well as to the poisonous effect of the faecal matter itself. When I saw the man in that collapsed, cyanosed state, it seemed to me that he really was being poisoned, not so much by the typhoid virus, as that had probably done its work, but by absorption of material from the contents of the intestine. With that in my mind, even when the diarrhoea was pronounced, I suggested that we should irrigate his colon. We, therefore, threw up into it a pint of warm water, and perhaps we ought to have had the courage to repeat it more than once. I cannot say that this single injection produced any marked amelioration, but when, at the post-mortem examination, one found that the large intestine particularly was full of semi-fluid faeculent material, concealing the large areas of ulcerated membrane beneath, it occurred to me that really the most rational method of treatment in such a case would have been a thorough flushing out of the diseased bowel. As we well know, that proceeding is very efficacious in ulcerative colitis which is due to causes other than typhoid fever.

Now as to the proofs of the suggestion that his collapsed condition and indeed his death were mainly due to poisoning from the intestine. The idea is not original by any means. One of the first to dwell upon the importance of the recognition of the fact of intestinal intoxication was the celebrated French physician, Bouchard, who has, indeed, been almost a pioneer on the subject; and it certainly appears to be the key to a great many disorders. This view of what is called self-poisoning (auto-intoxication) is that, even in the ordinary processes of intestinal digestion, there are a great many new chemical products formed, most

of which, if absorbed direct into the blood, would produce more or less deleterious effects. People who suffer from habitual constipation often present mental and physical symptoms (not of a grave order, it is true, but sufficiently irksome), which are entirely dispelled when the tendency to constipation is overcome. The late Sir Andrew Clark was convinced that many a case of chlorosis in young women was due to the fact that the intestines were not properly evacuated, and that the anæmic condition of this affection was due to this faecal intoxication. There are others who believe that graver forms of anæmia still may be brought about by the absorption of noxious products from the intestine. Bouchard applied this explanation to many other conditions. He based his view upon the discovery in the urine of substances, some of which could be isolated chemically and some physiologically, all having various poisonous effects on the organism when injected into veins or beneath skin. Even healthy urine, if introduced into the blood, is toxic. Poisons in the blood are presumably mostly eliminated by the kidneys, and if these organs are diseased the poison accumulates in the blood. Uræmia may be due, not to the accumulation of urea in the blood, but to toxic materials which have been absorbed into the blood, chiefly from the intestines. In typhoid fever, Bouchard himself says that besides the general poisoning due to the typhoid organism, one must pay attention to the local affection set up by it—this intestinal trouble, the necrotic lesions, embracing putrefaction of albuminoid substances. These products may themselves irritate the intestines, and cause diarrhoea, and, when absorbed into the blood, may lead to marked poisoning. Bouchard goes on to say that if a healthy person can be intoxicated by the products of digestion, how much more is this the case in such an abnormal condition as typhoid fever? This led him to insist on treatment being directed to neutralize these poisonous effects. Several remedies have been introduced for this purpose, which are known as intestinal antiseptics, such as carbolic acid, β -naphthol, salol, thymol. Some of these have been long prescribed in typhoid fever, but the original object of their employment was rather to neutralize the typhoid virus or toxine formed by the bacillus. We have no proof that they have this specific effect, but

there is proof that they do correct putrefactive changes, which must result from the continuous disintegration of tissue in the process of ulceration. No doubt, all cases of typhoidal diarrhoea are benefited by antiseptic treatment of this kind. Further, I would suggest, particularly in cases of obstinate and prolonged diarrhoea, such as occurred in the case under consideration, frequent flushing of the colon, knowing how beneficial it is in other forms of ulceration. So that the dangers to be borne in mind in enteric fever, as resulting more or less directly from the intestinal lesions, must include this self-intoxication; and summarizing the main conditions imperilling life in this disease, we have:—

First, the danger of death from the intensity of the fever process, from the poison acting upon the whole body through the blood. This cause, in years gone by, accounted for a greater mortality than any other, but since the teaching of Liebermeister and Brand in Germany, and I may say of Dr. Cayley in this country, it has been fully recognized that prolonged fever is itself a danger to life, therefore any means by which that fever can be reduced in intensity will save the body from the exhaustive effects of the fever, as well as from degeneration of heart-muscle, and so on, which before the days of antipyretic treatment used to carry off nearly one half the cases of typhoid fever.

Secondly, there are dangers due to the presence of particular lesions in the intestines. Knowing the liability to hæmorrhage and perforation, great care must be exercised in enjoining absolute rest and a very assimilable diet during the whole period the fever lasts. Though it may possibly be that the intensity of the infection is diminished by antipyretic treatment, it is difficult to attribute much neutralizing effect to it. However, some high authorities, such as Brand, of Stettin, consider that systematic cold bathing commenced early in an attack causes the intestinal lesions to abort, and therefore diminishes the risks arising from ulceration of the bowels, as well as those from the pyrexia.

Thirdly, there is the danger which I have been dwelling upon, of poisoning due to the absorption into the system of the products of decomposition in the intestine, of which I consider the fatal case we have been discussing an example; a danger which you will perceive arises after the other dangers may be said to have passed, when you are

beginning to hope that the patient is being safely moored in the harbour of convalescence. This toxic effect supervened in this man after four weeks of illness, and was, I believe, evidenced by the lowered temperature, the tendency to coma, and the cyanosis, conditions such as are observed towards the end of fatal chronic disorders of the intestines, and not strictly belonging to the "typhoid state."

Before concluding, I would like to touch briefly upon the diagnosis of typhoid fever in its early stages; time will not permit a full consideration, which I must postpone to a future occasion. We happen to have had at the hospital during the last month several cases presenting difficulties in diagnosis, some of which we failed to diagnose, for the reasons I will give. It is in truth almost inevitable for some mistakes to be made in the early diagnosis of typhoid fever, because there are no definite symptoms accompanying it. You know how very insidious is its onset, and most of the characteristic signs of the disease are often entirely absent during the whole of its course.

It is for this reason doubtless that the nosology has to admit into the lists the phrase—"simple continued fever," though of course such an expression is a confession of ignorance of the underlying cause of the attack. Now and then we meet with cases of obscure fever, which last a week and then subside, the onset having been insidious, and there is reason to think that such cases are to be set down to enteric fever which has aborted. There is no *à priori* reason why an intestine which is only slightly involved should not stop short at slight inflammation and everything return to the normal within a week. It is assumed by some authorities, I believe, that such rapid recovery from the effects of enteric virus may be brought about by the early administration of calomel; and therefore, in doubtful cases they give calomel to clear the intestine and prevent the infection from spreading. Some such cases are no doubt simply catarrh of the stomach and intestine, although many so-called gastric fevers are truly typhoid. If the temperature does not subside at the end of a week, one would naturally look for signs of the disease, including the characteristic eruption of rose spots. But these spots may, in a true case of typhoid fever, be conspicuous by their absence throughout the whole course of the

illness; diarrhoea may also be absent; or again, other symptoms may be wholly masked by the occurrence of some concomitants of typhoid fever. In illustration of that, I may mention the case of W. T., a man of 47, who is now in hospital. He came in on September 28th; saying he had never been ill before. On September 14th he felt some pains about him, and headache, and his doctor thought he had influenza. The pains and headache got better, but he suffered from cough, and on admission here presented marked signs of emphysema and bronchitis, the latter being of rather severe type. At first the case was doubtful; he was febrile (101.2° was the highest record), but there was always a morning remission. I had a suspicion of enteric fever, and we did detect a solitary, though equivocal, rose-spot on the skin of the abdomen, but as he seemed to improve he was put on fish diet, allowed to get up, and would soon have been discharged, cured of his bronchitis.

But on October 18th, after he had been getting up for nearly ten days, he had a sudden rise of temperature, and at the same time we learnt that his son had been taken ill almost simultaneously with himself, being sent to another hospital when the father came here; that the son also was convalescing, but had just had a relapse, and was put to bed again. They were, indeed, both cases of typhoid fever, and both behaved in the same way, in both, too, the real nature of the primary illness was overlooked, and it was the coincidence which gave us the clue to it. Bronchitis is often severe in enteric fever, and may mask other symptoms; a fact you will do well to keep in mind.

Another case in which we had a suspicion of typhoid fever from the beginning without positive proof, is that of a man of 59, whom I said earlier in the lecture had apparently contracted the fever at Margate. When he was there, in August, he was attacked with diarrhoea, and had severe headache. He came here a month later. He has had a long illness, but a febrile one. There have been some very imperfect spots, and a trace of albumen in his urine. He was placed on fever diet, and is now getting quite well, after some weeks' mild pyrexia without any definite enteric symptoms.

The next case I will mention possesses more interest still. A young maid-servant, K. P., came

with a history pointing to ulcer of the stomach. She was kept at rest in bed, and rapidly improved under treatment so far as her gastric symptoms went, but remained weak, and eighteen days after admission, viz., October 6th, her temperature rose from normal to 100° , 102° , then to 103° on October 9th. On one or two occasions it rose to 104° , and even 105° . In spite of that very marked fever, unassociated with any rigors, there has been no pain, and nothing to show that there has been any inflammatory lesion; nor were there any objective signs of typhoid fever, such as eruption or diarrhoea. The spleen, however, did become enlarged. The motions were natural, the tongue quite clean. However, her urine gives "Ehrlich's reaction," a reaction discovered by Professor Ehrlich, of Berlin—otherwise the "diazo-benzol sulphonic" reaction. This reaction consists in the appearance of a bright scarlet colour produced in the urine on the addition of liquor ammoniac to urine which has been previously mixed with a solution of sulphanilic acid (200 parts), nitric acid (10 parts), and nitrate of sodium (6 parts).

If this test were always obtained in typhoid fever, and if it never occurred in any other disease, then it would indeed be of the greatest possible value. But, unfortunately, we get the same test in measles; we have certainly often got it here in acute tuberculosis, and occasionally in other forms of disease, such as pulmonary phthisis; so that it is not pathognomonic. Then arises the question, "Is it ever absent in typhoid fever?" because, although its presence may not indicate typhoid fever, if it were absent we might be able to exclude typhoid fever. But some of the cases I have described to you would not have been typhoid fever if we had relied on this test alone. The test was yielded by the urine of the case I first related, but not by that of the other two male cases. It is, however, well marked in the case of this girl, K. P., but I confess I am not quite prepared to base a diagnosis solely on the fact. I am still of opinion that her fever may have another explanation. I am sorry to discourage you in the use of a test which is of a certain value, one of its valuable points being its presence in the early and most doubtful stages of the disease. Moreover, there is a fair consensus of opinion from various countries to the effect that the reaction is more frequently yielded by typhoid fever urine than by that of any other affection,

but others go so far as to say that it is of no value whatever, and that it depends on whether or not there is acetone in the urine (Von Jaksch).

NORTH-WEST LONDON CLINICAL SOCIETY.

ANNUAL MEETING.

An Address by the President,

Sir RICHARD QUAIN, Bart., F.R.S.,

BEING

SOME CLINICAL OBSERVATIONS AND PROFESSIONAL REMINISCENCES.

(By our own reporter.)

GENTLEMEN,—My good friend, Dr. Cagney, has, by some magnetic influence, induced me to address you here this evening. If I tell you that to-night I close my 79th year, you will feel almost as I do that I should be at home at my own fireside, but I have never allowed my own pleasure or convenience to interfere with what I felt to be a duty. I was, therefore, ready and very pleased to come to this clinical society, because I think what is practised in hospitals and discussed in clinical societies is the aim and object of our professional life—that is, the study and cure of disease.

I would desire to introduce myself to you by a few words of a personal character. In the year 1831 I became the pupil of an Irish surgeon-apothecary in Limerick. This was the year of the first invasion of cholera, and you can imagine that it was not a very encouraging experience for a youth fresh from school to come upon an epidemic of that kind. I well remember an early incident. My master had gone out in the morning, and an old gentleman, between seventy and eighty years of age, came and asked me to give him something, as his bowels were bad. I gave him the routine remedy at that time, it was "Ponsonby's drops"—a solution of camphor in spirits of wine, introduced by Lord Ponsonby as having been successfully used in Vienna—with the free use of iced water. A leading physician in Limerick at the time, named Lyons, when called to a case of cholera, invariably prescribed $\frac{1}{2}$ dram of calomel and some grains of

capsicum; and calomel in doses of from 5 to 20 grains every four or six hours were subsequently administered, until the symptoms abated or the patient died. I do not know that this treatment did more good or harm than Ponsonby's drops. About 75 per cent. of all the persons attacked died. On my master's return, being too fatigued to go out again immediately, he sent me to see him. I went and found that the old gentleman had died at six o'clock. This was a very great shock for a budding doctor, that a patient who had been able to come to him at twelve in the morning should have died at six o'clock the same evening. The disease was very virulent in Limerick at that time, but it was limited to a particular area. This brings me to a very interesting and remarkable point. Why is it that we now enjoy such immunity from cholera? Why is it prevented from invading our country? It is very much owing to the research and labour of a man whose name, I regret to say, is hardly known to many of you—Dr. Snow. He was the man to discover that cholera was propagated by polluted water. His conclusion was not accepted by such men as Dr. Bailey and Dr. Gull (afterwards Sir William) among others, in their Report to the Royal College of Physicians. However, a very bad invasion of cholera occurred in the neighbourhood of Golden Square, which was known as "the seizure of Golden Square district," and the Broad Street pump became a historical pump. Dr. Snow found that nearly all the people who drank water from this pump suffered from cholera, while those who consumed water from other sources escaped. An old lady and her niece migrated to Hampstead, and they died of cholera there, the only cases in that neighbourhood. Dr. Snow showed that these good people had had water sent to them daily from their favourite pump in Broad Street to Hampstead. Nothing could be so strikingly confirmatory of Dr. Snow's views than this as to the propagation of cholera by polluted water. This doctrine was fully substantiated by Mr. (Sir John) Simon (who gave credit to Snow for his zeal and judgment) and his distinguished aids, Dr. Parkes and Dr. Netten Radcliffe. Yet, is it possible that probably not more than nine-tenths of the people now living know to whom this great discovery is due? There is no memorial to him, no monument has been raised to commemorate his name and work. But I hope this oppro-

brium will not be allowed to continue, and that we shall see such a memorial as a granite monument, perhaps, on the Thames Embankment, recording the fact that Snow was instrumental in saving millions and millions of lives. Snow lived in Savile Row, but did not reach 50 years of age. He was known for his writings on chloroform and other anæsthetics. Had he been a general in the army he might have received a peerage and a pension, having been instrumental in destroying the lives of thousands. Snow saved the lives of millions, and is forgotten. This should not be.

Now, gentlemen, no one can doubt the value of clinical societies and the work they do. Without stopping to inquire in what special ways they are valuable, I would accept their extension as proof of their recognized value, and would mention that in my early days in London there were only four societies—the Medical, the Hunterian, the Medico-Chirurgical and the Westminster, whereas there are now twenty at least, not including those connected with medical schools. The extension of these societies proves their value, otherwise they would not exist. I am under the impression that the duplication and reduplication of some of the special societies dilute and diminish their value. It has been suggested that the Medico-Chirurgical should be a great centre for all the societies. I have in my possession a set of rules and regulations which were framed under the guidance of Sir George Burrows and Sir H. Pitman, with the object of converting the Medico-Chirurgical Society into the Royal Medical Society of London. The proposal did not succeed, and we are all now very much divided. The Pathological Society was the first branch from the Medico-Chirurgical. It was then suggested that there should be pathological evenings at the Medico-Chirurgical, but the suggestion failed. However, the Pathological Society was founded, and it has had great success. I had the privilege of being one of the founders, but Dr. Bentley was the actual founder. This is another name I would like to rescue from oblivion. He was a Guy's Hospital man, and saw what scope there was in London for such a society on similar lines to the Dublin Pathological Society. He applied to a number of physicians and others, and their adherence having been obtained, the Society was started. Dr. Bentley also founded the Victoria Park Hospital for Diseases of the Chest, and it is

painful to realize the fact that the names of Snow and Bentley are practically unknown.

Well, gentlemen, I do not know of anything more important than the study and concentration of the several branches of our science, which are pursued with so much zeal in the treatment and cure of disease in the wards of a hospital. Valuable as all these researches are, we must not lose sight of the ultimate object, the prevention and cure of disease. I was reading the other day a most interesting address on botanical progress by Dr. Thistleton-Dyer. He says:—

“The modern university student of botany puts his elders to the blush by his minute knowledge of some small point in vegetable histology. But he can tell you little of the contents of a country hedge-row, and if you put an unfamiliar plant in his hands he is pretty much at a loss how to set about recognizing its affinities.”

The wards of a clinical hospital may resemble the hedge-row. Those who devote themselves exclusively to minute researches may possibly feel as much at a loss in rows of beds in the wards of a clinical hospital as the man did in the hedge-row of Dr. Thistleton-Dyer. I therefore would urge upon you, above all things, the study of every and all the phases of disease. It is very well to learn from the labours of those devoted men who work out these subjects, it may be in anatomy, physiology, histology, and morbid anatomy, but you must seek to apply this knowledge practically. We are called upon to cure disease; it is our whole aim, if we cannot prevent its occurrence. And nothing conduces to that object more than a clinical hospital, where you can study the phases of disease and bring your knowledge of every department of our science to bear in its treatment. I therefore heartily wish success to your hospital and your clinical society.

Now, I hardly know how best to bring before you the facts of some cases which may be of clinical interest to you, and in the course of a long professional life I have met with some very remarkable cases. I would like particularly to refer to some cases in which albumin was a constituent of the urine. Nothing could have been more unfortunate than that the name “Bright's disease” should have been given indiscriminately to the mere presence of albumin in the urine. It scares everybody, alike the patient and the

doctor; they imagine that there is or must be some structural disease of the kidney; that this is "Bright's disease;" that it is a specific malady; and that you can do very little for it. Believe me, that is a great mistake. I will give some illustrations. I had brought before me a gentleman, a very active literary worker, and whose whole life was engrossed in work. He lived generously and well, and the malady he came to me for was the presence of albumen in his urine. The specific gravity was 1015, and there was occasionally a hyaline cast. I recommended him to get his liver right (it was large and congested) by small doses of calomel; and from this he felt so much benefit that he kept a little ivory box in his pocket containing pills of $\frac{1}{4}$ to 1 gr. calomel, and whenever his digestion seemed disturbed, down went a pill. He continued in very active employment for twenty-five or twenty-six years after that. He had, at one time, discovered that there was, around the corona glandis, a ring as hard as of iron wire. There was no excoriation, and there were no swelled glands. I asked my friend, Mr. Henry Lee, to see him with me. He did so, and said, "I don't know what to make of it; there it is. I think he will be better treated as if he had syphilis." We agreed to give him some blue pill every night. He was a man of enormous energy, and very self-willed, and we found, after two or three days, that he was taking 25 grains of blue pill a day. In addition, he had some mercurial inunction, and he was so eager to get rid of "this filthy thing," as he called it, that he exposed his penis to mercurial fumigation. He got rid of "this thing," and his albumin was not affected in the slightest degree.

"Well," said Mr. Lee, "I didn't think it would, because I remember one case where I gave a man mercurial vapour bath, with the effect of diminishing his albumin."

There is a morbid feeling as to the impropriety of giving mercury in cases of albuminous urine. I am sure, however, that mercury in some form or other, combined with squill and digitalis, is invaluable in certain cases of albuminous urine. I believe it relieves the congestion in the abdominal organs, including the kidneys.

The case I have narrated is not an exceptional one. I was consulted by the parents of a young lady, 18 years of age, who, when menstruating, went into

the sea to bathe. This stopped the menstruation, and the result was general oedema. That happened in the month of May, and I saw her in the following November. She then had copious albuminous urine, and oedema all over the body; in fact, her face was so puffed that she could hardly open her eyes. I recommended hot-air baths, with salines, etc., but they had no effect. At last I gave her 3 grains of Plummer's pill, which contains about $\frac{2}{3}$ grain of calomel, three times a week. In addition she took citrate of iron daily. By degrees all the oedema disappeared, and the albumin in the urine became much reduced (there were no casts), but it never quite left her. However, she got well. I should say she took 3 grains of Plummer's pill 3 times a week for a year. After being under observation more or less for eight years, she went to the Cape of Good Hope, and when she returned became engaged to be married. As a curiosity I show you this lady's portrait, which she gave me just before she married. Unfortunately, after two years of married life she got congestion of the lungs during influenza, and died.

I want to impress upon you that, under certain circumstances and conditions, mercury is a most useful remedy in certain forms of albuminous urine, and that it should not be avoided. The origin of the objection to it may be found in the work of Dr. Blackall, of Exeter, who, in 1820, wrote on dropsy connected with kidney disease. If you read this paper side by side with Dr. Bright's, you will see how nearly Dr. Blackall anticipated the distinguished physician after whom the disease is named.

But, gentlemen, after all, it is very difficult to say what "Bright's disease" is. On one occasion an Irish lady, who was suffering from heart disease, congestion of the kidneys, and albuminous urine, was seen by Sir William Gull. The consultation was brought about by the fact that her doctor, Frederick Weber, had let drop, in the patient's presence, the words "Bright's disease," whereupon the lady exclaimed, "What! have I Bright's disease? I must ask Sir William Gull."

We told him the facts, and when he saw the lady, she said, "Now, Sir William, have I got Bright's disease, or not?" "Well, madam," said Sir William, "what is 'Bright's disease'?"

He tactfully passed the point away in conversa-

tion, and when he had gone she exclaimed, "But he did not tell me. He asked me, and how could I know?" I said, "No, he does not know, neither do you."

Speaking of Dr. Bright reminds me that I had an opportunity when sitting next to him in one of the earliest meetings of the Pathological Society, and I happened to have a drawing, which I present to you, showing the two most opposite forms of kidney disease. I said, "Dr. Bright, which of these is Bright's disease?" He replied smilingly, "I will take them both." I have presented these almost historical drawings to the College of Physicians.

It has been unfortunate that the name "Bright's disease" should have been used in that way, because there is no doubt that very many cases which are so called present none of the features of structural disease of the kidneys. I will give you an instance.

A lady of rank said to me, "I wish you would look at my boy." He was the only son, and heir to a very large fortune. I said, "Bring him," and she did so. He was almost a skeleton, a wretched little fellow, 6 or 7 years old. I could find no evidence of organic disease. I examined his urine, and found sugar and albumen in it, and this I told his mother. In the afternoon of the same day her husband came, and said, "You say my boy has got sugar in his urine. Could that have arisen from his eating sugar?" He went on to say that Sir William Gull had been consulted, and told that the boy was constipated. "Oh!" said Sir William, "let him eat plenty of bread and brown sugar." He indulged immoderately in this diet, the result of which was that he had albumen and sugar in his urine. Of course the bread and sugar were stopped, and the boy got well, or at all events stronger and better. I lost sight of him for five or six years, and then he was brought to me by his father's butler, who had brought him from Eton.

"Eton," he said, "is broken up with scarlet fever, therefore master desired me to bring the boy to see you; he has a sore throat, and is not very well."

On examination, I found a large quantity of albumen in his urine; his throat was a little sore, he had no rash, but he had a very large abdomen.

He said, "I have been eating too much." It

transpired that he had two baskets sent him each week, and two other boys in the same house also had two each, and with the pies, tarts, and all that sort of thing, the poor boy had got a tremendous liver.

His father telegraphed, "Send him home; we have a very good doctor here." He went across to Ireland accordingly, but the father's doctor said, "Take him back again, I will have nothing to do with him." He was therefore sent back, and I looked after him for a few days.

Shortly afterwards, when riding in Rotten Row, he was thrown from his horse. He was taken to St. George's Hospital, and placed under the care of Mr. Dent and Mr. W. Rivers Pollock, who was then acting house-surgeon. Mr. Dent reported that the boy had a fracture of the orbital plate. There was hæmorrhage round the eyes. His parents were, of course, much alarmed, and bearing in mind his albuminous urine, we thought it a very serious case. The youth was there treated with milk and beef tea, etc., and his albumin, which was formerly very evident, gradually declined and almost disappeared. He was then transferred to the hotel where his father and mother were staying, and when he returned to the ordinary fare of healthy people the albumin reappeared.

So anxious were his parents, that I was supplied with a specimen of his urine three times a day, and each time it was accompanied by a bill of preceding fare, so that I should trace what food suited him. I may mention, parenthetically, that cold chicken and tongue caused the largest amount of albumin in his urine.

The next I heard of the young gentleman was that he was at Cambridge, where he was fortunate enough to come again under the care of Mr. Pollock, who was at Addenbroke's Hospital. At Cambridge the youth became master of the harriers. He has grown into a healthy young man now, is married, and has lost all trace of albumin in his urine. So that albuminous urine, however much and however recurrent, does not mean serious and incurable "Bright's disease." The care of diet, purgation, and reduction of his liver did infinite good.

This brings me to speak of the liver, as an important subject, and a thing clinicians ought to take special note of. We must remember as

proof of the importance of the organ, that every animal in creation, from the highest to the lowest, has a liver, or something acting as such, therefore it must be an organ of very great importance. I believe sufficient notice is not taken of it. I wish I were sufficiently skilled in organic chemistry to trace all the sequences of sugar, albumin, and urea in the urine; that is not in my power, but I can give you an illustration of liver trouble influencing the presence of urea in the urine. It was formerly said that however much urea you may find in the urine, and whatever the specific gravity, that it depended on the quantity of urine which is passed being limited.

I was once consulted by a gentleman, under forty, living in the Temple, whom I saw with Mr. Savory, afterwards Sir William. Mr. Savory said to me, "Look here, that fellow is dying, but I don't know what is the matter with him." He was a good-looking jolly, steady fellow, and said to me, "I can not do anything, doctor; the fact is I am no use." I said, "Are you very weak?" "Yes; I don't care to go out, and I don't care to do anything." I said, "What do you eat?" "Well," he said, "I like vegetables, which are a good part of my food, and I take very little stimulant." I said to Mr. Savory, "Surely we ought to make out, between us, what is the matter with him. How is his urine?" "Oh," said Mr. Savory, "it is all right." "Well," I said, "I would like to see some of it." I had a specimen, and found the specific gravity to be 1035; it was loaded with an excess of urea. Next time I met Savory I mentioned this to him. "Oh," he said, "I suppose he passes very little urine." But on inquiry, we found that the patient passed from six to eight pints daily. He had a large liver, and this led me to feel how much the liver had to do with this excretion of urea. A French physician, Dr. A. Martin, wrote (Paris, 1877), pointing out that the variations in the amount of urea in the urine may often be used in the diagnosis of diseases of the liver, and I have myself found that this is correct. Many persons come to a physician and say, "I am so depressed, so languid, that I can do nothing." If you examine their urine you will very often find excess of urea. Put the liver right, and you will do wonders in alleviating the symptoms. The late Mr. Carlile, a man of recognized power of judgment and discrimination, who lived to eighty-

two years of age, would not take any medicine but grey powder. He was subject to dyspepsia, a fact which may be explained by his fondness for nasty gingerbread. I have often seen him sitting in the chimney corner, smoking a clay pipe and eating this gingerbread; but he overcame its ill effects by the grey powder.

I will now pass on to tell you of some curious cases I have met with regarding morbid impressions.

I was asked, on one occasion, to see a gentleman in Harrington Square (not far from here), with my friend Mr. Claremont, of Hampstead Road. He said, "This is a very curious case. The man is an eminent solicitor, and he has a morbid impression that there is an offensive smell about him. He goes to the office and transacts business with his partners; he hides the fact from them, but he won't hide it from you." I saw him in his bedroom; he was dressed most neatly, and everything was specially clean. He said, "I am very sorry to bring you here, my smell must be very bad for you; I am afraid your horses will feel it at the door." Even the pictures in the dining-room were covered up to preserve them from the offensive matter. I felt his abdomen, and when I got to his right iliac region I found a faecal accumulation, and called Mr. Claremont's attention to it. The gentleman immediately said, "All the smell comes from there." It was autumn, and I went away from town. When I returned I saw Mr. Claremont, and he told me he had opened a caecal abscess, and that nothing could be more intolerable than the stench of it. The patient got well. Is it possible to account for that? Did the offensive material get into the blood and so affect the olfactory nerves? At any rate the man was cured by the discharge of this caecal abscess.

The second case is not quite so remarkable. I was asked by Dr. Walsh to see a gentleman of rather eccentric habits, who lived in Upper Baker Street. When I went to see him, the people with whom he lodged said he had been shut in his room for three or four days. When the door was broken open, he was found lying in bed, quite insensible, and there were the remains of a mutton chop which he had given his favourite parrot, his only friend. He had always spoken of a "block" from which he suffered: he would not eat, and finally this block he said forbade him eating at all. We agreed that

something must be done for him; we brought him to consciousness, but he died in the night. The post-mortem revealed a lump of fæces, the size of a closed fist, in his rectum, as hard as iron. The patient had evidently starved himself to death in consequence of the block.

I would now like to mention a case which shows how perseverance in treatment may be successful under most unfavourable circumstances. It was that of the late Lord A., we will call him. He was sixty-three years of age, of great intelligence, and of most amiable disposition. He had been a patient of Dr. Lombard, of Geneva, he who was the first physician to indicate the difference between typhoid and typhus fever. Lord A. being at college at Geneva had acute rheumatism and endocarditis. Many years after that he came under my notice. It was in the month of November, and he was suffering from a mild form of influenza, during one of those fogs for which we are so conspicuous. He was anxious to get away to the South of France, but wished to see Drs. Bence Jones and Watson first. Bence Jones remarked to me, "The man ought to have died long ago, and that he had told him so;" and whenever Lord A. met him he assured him good-humouredly that he was not shaking hands with a ghost. Lord A. went to Paris, and was accompanied by Dr. David Christison, son of Sir Robert Christison. On the third day after his departure I got a telegram, "Lord A. is very ill; pray come to-night." I went, and arrived at Paris the next morning. I met Dr. Christison (who now lives in Edinburgh). He said, "His lordship has got congestion at the base of his left lung." I knew that at the time he was suffering from albuminous urine, and disease of the mitral valve. He had a systolic bruit, and some enlargement of the heart. He was very low. Nothing could look more unfavourable. Christison said, "Her ladyship yesterday sent for Sir Joseph Olliffe, and we have had Baron Louis and Dr. Trousseau in consultation. They prescribed grey powder and some antimonial powder. Dr. Trousseau being asked about his diet, said, 'Give him ptisan; nothing else.' 'Not beef-tea?' asked her ladyship. 'No, no!' Seeing that this was altogether contrary to the principles I had laid down for the treatment of the invalid, I was telegraphed for. On seeing the patient and his condition I immediately procured

from the English chemist a supply of *Mistura Spiritus vini Gallici*. I believe this to be an invaluable remedy in like cases. Then I had a blister applied over the seat of the pneumonia. M. Trousseau arrived between ten and eleven, and Baron Louis shortly after. I explained that I felt it necessary to give him a stimulant. He, M. Trousseau, said, "Do you cure pneumonia in London by *eau de vie*?" I said, "No, but I should desire to keep his lordship alive while your remedies are doing him good." Next time M. Trousseau came (on the first occasion he made a long speech) his lordship feigned sleep and would not notice him. The visits of the French physician, who gave no hope, were discontinued. Sir Joseph Olliffe and Dr. Christison took charge of the case. There was some appearance of disease at the base of the right lung. Though between sixty-three and sixty-four years of age, the patient got slowly better, and after a stay of seven or eight days I returned home. Then that excellent physician Baron Louis was requested to come to see the patient, and said he was rejoiced at the result. He thought that his lordship might be tired of brandy. He said he knew there was some excellent Burgundy in the Hotel Bristol, and it was given him. In three days the kidneys stopped secreting, and the body swelled generally with anasarca. I was again sent for and gave blue pill and squill, &c. Louis also was sent for again. He said, "This is very sad." And I replied, "Either it is the effect of the Burgundy, or of suppressed gout." "No," he said, "it is over-feeding; you have given him so much to eat. But what will you do?" I said, "We will give him blue pill." He said, "Do you cure Bright's disease in London with blue pill?" I replied, "We use it with success in like cases." Fortunately the blue pill was quite effective; the urine came and the dropsy went, and he got much better. Towards the end he became extremely weak; and he got aphthæ, and his mouth and throat were very sore. I got many more than a hundred telegrams about this case, and I crossed to Paris seventeen times during the period between November and June. The telegrams must have cost a fortune in those times. He got better, but the aphthæ remained for a time, and were very painful. "Quain," he said one day, "this is horrid; this thing has been going on badly for two days; if it continues I

can't swallow after to-morrow. If you can't stop it I shall die, and there will be an end of it." It was stopped, but he was so exhausted by an attack of gout which then developed itself that he got a very sore back. Notwithstanding that he had a water-bed the sacrum was exposed. Yet he got better, and removed into a healthful house in the Champs Elysées, finally coming home, and going to his country seat in July. He went on extremely well during the remainder of the summer, and rode on horseback. However, in driving out late on a November evening he got cold, and had pleurisy. We got him through this, but eventually he died suddenly of the heart disease in March. He might have lived much longer if he had not driven out when other people were coddling round the fireside. M. Trousseau was so impressed with this case that he introduced the subject in one of his clinical lectures, winding up thus, "Well, gentlemen, had he been a Frenchman he must have died."

I will now mention to you a case or two of another kind. I was required by Mr. Wakley, the coroner, to make a post-mortem examination (and he asked me to make many) of a man whose story was this:—A stonemason, he came in to his dinner, then went into the scullery to wash his hands. On returning he said to his wife, "There; it is all over; I have taken poison; I have taken arsenic." It was near the Western General Dispensary, of which Dr. Humby, who subsequently had a large practice in Wellington Terrace, was then the resident medical officer. He was out, but two young pupils were there, very good fellows, I have no doubt, and well-meaning. They naturally thought it a very grave case, and they treated it pretty actively. They gave him tartar emetic, and oxide of iron. He was extremely ill, had vomiting and diarrhoea, and died in twenty-four hours. The coroner's beadle said to the chemist, "How did you come to sell this man poison?" He said, "I sold him no poison. I saw he was off his head, so I gave him some alum and cream of tartar, and labelled it poison." On examining the body I found a great deal of arsenic in the stomach, but there was none in the duodenum; and there is no doubt these young men were frightened when the man died, and pumped some arsenic into the stomach, where I found it. Mr. Wakley was a very stern man, but withal one of the most amiable and excellent

men I knew. He said, "I am not sure that those fellows are not guilty of manslaughter; I will commit them." I said, "For God's sake don't do that; I am sure what they did was well-intentioned." I persuaded him to overlook it all, and condone the contempt of Court, as he said, in practising a deception. I was very glad, because it would have been a terrible blow to these young gentlemen.

I have occupied a great deal of your time; but, as President of the Medical Council, you will excuse me saying a few words about that much-abused body. I have been a member of the Medical Council now more than thirty years, and my conviction is that body will by-and-by have enormous influence for good on the medical profession. I remember when political reform was in the air there was also a great cry about "medical reform," and the cries of that time were—(a) the profession to govern itself, and (b) uniformity of qualification, and reciprocity of practice. At that time people educated in Scotland and Ireland were not qualified to practise in England, and *vice versa*, but though uniformity of qualification has hardly been reached, reciprocity of practice has long been fully established. Then and subsequently, many appeals were made to Governments for "medical reform"; some people wanted much, others little. One Government proposed to itself appoint a medical council, and to control the registration, that is to say, to take the control of the medical profession entirely out of its own hands, and that the Registration fee should be paid as a stamp tax to the Government. However, that was very much opposed. The beginning of this was that, as early as the year 1831 or 1832 there was a Board, called the Board of Health, appointed to resist the invasion of cholera. This board subsequently became the general Board of Health, and at that time (1854-5) Sir Benjamin Hall, a man of radical tendencies, and a homœopath, was President of it. A deputation went to ask him to give us a medical bill. He said, "What do you want? Do you desire to tell me that I may not consult anybody I like?" "Yes, we said, you may go to the greengrocer if you please, but we won't allow him to be called Dr. Green." He assented to that, and added, "Let me remind you that when Lord Althorp was Chancellor of the Exchequer he told the House

of Commons a story *à propos* of this question, of no one being allowed to prescribe who was not qualified. He said, 'the last financial statement I had to make was one day when I suffered very much from gout on leaving home, and I was inclined to turn back, but instead of that I went to a chemist in Parliament Street, who gave me some medicine that did me good immediately. Am I not to be allowed to seek relief in this way?' That tradition got hold in the House of Commons." You must not prevent people from prescribing, but you will prevent them calling themselves doctors, or by any deceptive name, and thus imposing on the public.

Now, in reference to the Medical Council, I ask you to look at the very distinguished men who have been on that Council, and who have taken a share in its work—originally Brodie, Green, Watson, Burrows, Christison, Syme, Stokes, Corrigan, and a number of men, their successors, of the highest character; such men have always meant well for the interests of the medical profession—it is inconceivable that they should have done otherwise—but they have been prevented from doing all the good they might have done by jealousies, rivalries and misunderstandings.

By degrees, however, the present greatly improved state of affairs has grown up, not by legislation, but by moral suasion, as Mr. Syme called it. I believe all the bodies are coming round to see that it is most desirable, in the interests of the profession, that if there is to be rivalry it should be a rivalry in doing what is right. I believe that in a little time, under the influence and guidance of the General Medical Council, by not proceeding sternly or rigidly, a great advance will be made in the education of the medical profession. When I first entered the profession, the examination of the College of Surgeons lasted one hour—half for anatomy, and half for surgery; now the joint examination extends over thirty-two hours. Two and a half to three years was the curriculum, now it is five years. Very much of this improvement is due to the Medical Council, representing, as it does, all the licensing authorities. You really have the government of the profession in your own hands. Solicitors have no such power as we possess: they submit to heavy taxation, they pay 105% on admittance as solicitors in stamps, and some nine pounds or so annually. However "infamous" a

member of that profession may be, he must be brought before a Court of Law. If we declare the conduct of a member of our profession infamous, there is no appeal, and this makes us extremely careful in our judgments. There are degrees of disgrace which are less than infamy, and which ought to be corrected. This should be done under the bye-laws of the several medical authorities. At this moment I have had a case brought before me of a Liverpool grocer who gave away to purchasers of his tea a ticket entitling them to advice and attendance from a choice of three doctors. Can any greater prostitution of our profession be conceived than that such persons should be able to hire themselves out as bribes to the buyers of tea?

I have talked longer, gentlemen, than I intended, and I thank you very much for the attention you have given me. (Great applause.)

ALCOHOLIC DELIRIUM.

(D.T. and T.D.)

By SAMUEL WEST, M.D., F.R.C.P.,

Assistant Physician to St. Bartholomew's Hospital;
Senior Physician to the Royal Free Hospital.

I do not propose to enter into any description of alcoholic delirium. It is a condition, unfortunately, only too familiar to us all, and one that presents but little difficulty in diagnosis. The general tremulousness of the patient, coupled with the busy, active delirium with hallucinations, is so characteristic that there is little to confuse it with, unless it be some peculiar form of acute mania and some rare cases of poisoning; but the condition of the patient, the history and course of the disease render confusion very unlikely.

There are two forms of alcoholic delirium which ought to be kept distinct from one another, for though in both the delirium and tremulousness are the same and the cause is the same, viz. the abuse of alcohol, still in nearly every other respect they differ, for they differ in regard of history, course, prognosis, and treatment. To emphasize the distinction I have been accustomed to speak of the one as "D. T." and of the other "T. D.," that is to say, Delirium Tremens and Traumatic Delirium.

In "D. T." the patient is a chronic drunkard, has been constantly drunk until the attack came on; in other words, has drunk himself into the fit. While in the other ("T. D.") the patient, though an alcoholic, may never, or hardly, ever, have been drunk, may be a respectable member of society, and capable of good work, and would never have had an attack of delirium tremens if it had not been for a fall, an injury, an accident, some fever, or severe illness. Thus, I recall the case of a coachman, an old and trusted servant of a family for many years, who was knocked down in the street and fractured his thigh. The same night he developed the symptoms of delirium tremens, took off his splints, got up and walked about on his broken leg, and converted a simple into a compound fracture, and in two days' time he died. Although it turned out that he was a free drinker, he had never been in his life, so far as could be ascertained, incapacitated from work by drink.

I have seen typhus, as well as pneumonia, diagnosed as delirium tremens, and although the temperature was high, it was not thought to be of importance in the diagnosis, which was not made, in the first case, until the rash appeared, and in the second at the post-mortem examination.

If sufficient stress had been laid upon the temperature this error in diagnosis would, in all probability, have been avoided, for although there certainly are cases of simple delirium tremens in which the temperature is raised, and in the rare fatal cases it may possibly rise to a considerable height just before death—still, speaking generally, the temperature is low; and I think it is a good general rule to lay down that if, in a case which appears to be delirium tremens, the temperature be high, the suspicion should at once be roused that delirium tremens is not the whole of the disease, but that it is an accident, or a complication of some other more serious affection.

I have already said that these two groups of cases differ from one another in a very marked degree.

D.T.—The patient who has been drinking up to the time of his attack, has a moist skin, a moist and coated tongue, a soft pulse, of low tension and no great rapidity. These cases are best treated without narcotics or stimulants. For the first day or two, while the tongue is coated and

there is little sleep, they should be fed on liquid food, but plenty of it, and as soon as the tongue cleans and the appetite returns, that is, in a couple of days or so, they should be put at once on solid food.

Stimulants, even in small amounts, aggravate the symptoms and retard recovery. I have never seen any harm come of cutting off all stimulants at once, in fact, nothing but good, for it hastens convalescence. Nor are narcotics necessary. In the first place, if the stimulants are cut off, it will be but two or three days before they sleep naturally, and in most cases there is little doubt that their strength will be sufficient to hold out for this time. Secondly, if narcotics are given, they would have to be given in very large amounts, and by producing general disturbance would impair appetite, produce constipation, and render the general condition worse.

The prognosis in these cases is almost invariably good, so that in less than a week the patient is on the high road to convalescence.

In T.D., on the other hand, the patient gets the attack as a consequence of some accident or illness. There is often no history of drunkenness or of previous D.T. The symptoms develop twenty-four hours or so after the accident or commencement of the illness; the temperature is usually raised, and sometimes reaches a considerable height. The skin is dry, the tongue dry, the pulse rapid, often hard, and the tension high.

These cases are associated with alarming asthenia; for though the patient may require two or three attendants to keep him in bed, still his muscular activity and mental excitement are out of all proportion to his real illness; the condition of his heart and pulse show how rapidly the asthenia increases, and soon he lies prostrate in the bed exhausted, and dies in a condition of collapse. To combat this asthenia stimulants are necessary, and often in large amount. In the same way sedatives or narcotics are required to procure sleep if possible, and where the fever is high a cold douche to the head or general sponging of the body may tend to soothe the patient to some extent.

The prognosis is very bad, for a large number of these cases are fatal, and under any circumstances convalescence is slow.

The difficulties in diagnosis are not, as a rule,

great, for there is the history of the illness or accident to assist the diagnosis, and the delirium, in most cases, is clearly secondary.

The difficulties are greatest in the cases of illness, rather than of accident, as, for instance, in pneumonia, where the onset of the disease may have been marked rather by the delirium than by any of the other signs. These are the cases in which mistake is usually made, but if attention be given to the temperature and other signs of fever, error is hardly likely to occur. It is a good general rule to lay down that a case of apparent D.T. with a high temperature is very likely to prove to belong rather to the other group, viz. that of Traumatic Delirium.

Considering how opposed these two groups are to one another, both in the treatment which is necessary and the prognosis of the case, it is clearly of great importance that the character of the case should be recognized at once.

THERAPEUTICAL NOTES.

Scrotal Pruritus.—*Carbolic acid*, 20 parts; *neutral glycerin*, 75 parts; *alcohol*, 25 parts; *distilled water*, 300 parts. Make a mixture of 1 part of this solution and 4 parts of very hot water; dip eight or ten layers of tarlatan in it and apply as a compress, completely enveloping the scrotum, keeping it in place by a rubber suspensory. Give as a sedative, *carbolic acid*, $\frac{7}{8}$ grain; *extract of valerian*, $1\frac{3}{4}$ grains; *powder of valerian*, 3 grains. To make one pill. From 3 to $9\frac{1}{4}$ grains to be given daily at meal-time. Prohibit all stimulants, and in severe cases order exclusive milk diet.

(*La Presse Méd.*)

Chloralose as an Hypnotic.—Marandon de Montyel, from a study of fifty-eight cases in which he employed this drug as an hypnotic, condemns its use in insanity, general paralysis, and senile dementia, and warmly recommends it in epilepsy. (*Rev. de Méd.*). Féré reported excellent results a year ago from its use in this class of cases, and the observations of the present author confirm his assertions. It is to be noted, however,

that patients who sleep well and whose attacks of mania are notably shortened under its influence, seem at the same time to be very susceptible to its physiological action; this is especially true of epileptics, who show extreme sensibility both to its favourable and unfavourable effects.

Bismuth in Ulcerative Gastric Affections.

—Wash out the stomach in the usual way with a solution of *bicarbonate of soda*, and introduce through a stomach-tube $2\frac{1}{2}$ to 3 drachms of *bismuth subnitrate* in fifteen ounces of water, shaking this mixture continually to prevent its settling. Let it remain ten minutes in the stomach, meanwhile removing the tube. Then carefully withdraw the water, which should come away clear, leaving the *bismuth* deposited on the inflamed and ulcerated membrane of the stomach. Most efficacious in chronic ulcerative gastritis from alcoholism and in simple round ulcer of chlorotic girls.

(FOURRIER, *La Presse Méd.*)

Glycerophosphates.—Indications: Breaking down or depreciation of the nervous system, as in convalescence from acute diseases, especially influenza, in phosphaturia and the neurasthenia following it, ataxic pains, tic douloureux, sciatica, chronic lumbago, and Addison's disease. Favourite formulæ of author: *Glycerophosphate of lime*, $1\frac{1}{2}$ drachms; *glycerophosphates of sodium, potassium, and magnesium*, each 31 grains; *glycerophosphate of iron*, $15\frac{1}{2}$ grains; *tincture of St. Ignatius's bean*, 30 drops; *pepsin*, 46 grains; *maltine*, $15\frac{1}{2}$ grains; *tincture of kola*, $2\frac{1}{2}$ fluid drachms; *cherry-syrup*, $6\frac{1}{2}$ fluid ounces. A tablespoonful during midday and evening meal. Or the following cachet, given at meal-time: *Glycerophosphate of lime*, $4\frac{1}{2}$ grains; *glycer phosphates of sodium, potassium, and magnesium*, each $1\frac{1}{2}$ grains; *glycerophosphate of iron*, $\frac{7}{8}$ grain; *powder of St. Ignatius's bean*, $\frac{1}{2}$ grain; *pepsin*, $2\frac{1}{4}$ grains; *maltine*, $\frac{7}{8}$ grain. For the treatment of chlorosis and anæmia; *Glycerophosphate of iron*, $\frac{7}{8}$ to $1\frac{3}{4}$ grains; *rhubarb powder*, $\frac{1}{8}$ grain; *extract of quinquina*, $2\frac{1}{4}$ grains. To make one pill. Two or three to be given daily at meal-time.

(ALBERT ROBIN, *Bull. Gén. de Thé.*)

THE CLINICAL JOURNAL.

WEDNESDAY, NOVEMBER 27, 1895.

TWO CLINICAL LECTURES

ON

ENTERIC FEVER.

Delivered at the Middlesex Hospital, on October 26th, and November 15th, 1895.

By **SIDNEY COUPLAND, M.D., F.R.C.P.,**

Physician to the Hospital.

LECTURE II.

ANOMALOUS TYPES OF ENTERIC FEVER.—
RELAPSING CASES.—“ICE-CRADLING.”

GENTLEMEN,—You may remember that towards the close of my last clinical lecture, which was mainly concerned with a remarkable instance of extensive ulceration of the colon in enteric fever, I briefly referred to two or three other cases then in the wards as showing some of the difficulties met with in the diagnosis of this affection. As three weeks have since elapsed it may interest you to learn something of the subsequent progress of these cases, which will, moreover, give me an opportunity of further illustrating some of the features of enteric fever, which has been very prevalent this autumn. As my remarks will be confined to cases which we have recently had in the wards, they may appear to be somewhat desultory—but I have no desire to attempt a complete and systematic description of the disease, which you have already doubtless received in another place from one of the highest living authorities upon it.

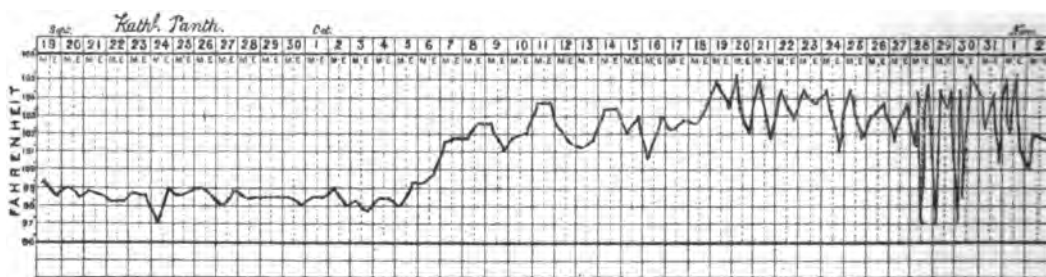
In many instances, from the lack of any of its distinctive signs, a diagnosis of enteric fever can only be arrived at by a process of exclusion. The case of the young woman, K. P., to which I referred last time, belongs to this category. She was admitted into the hospital on September 19th, complaining of epigastric pain after taking food, and of vomiting about once daily during the past three weeks. She had come from Hendon about six weeks ago to enter domestic service in London, and during nearly the whole of this time she had been ailing with these apparent dyspeptic troubles,

having on two occasions “brought up” a little blood. The pain was relieved by vomiting; but she became so obviously unfitted for work that a few weeks’ hospital rest and regimen seemed requisite to restore her to health. She was a pale brunette, dull and listless in manner, having a coated tongue, and being notably tender on palpation of the epigastrium. There was occasional vomiting and marked constipation. The skin was cool, temperature not exceeding 99°, usually normal. Pulse 90, fairly full, and compressible; heart and lung signs normal. With the history of these symptoms, one felt justified in making a diagnosis of simple ulcer of the stomach with anæmia—a common combination; and the treatment was based on this view of the case. Milk diet, a bismuth mixture, and occasional doses of cascara to relieve the tendency to constipation led to marked improvement in the gastric condition; the vomiting, which had become variable in its occurrence, ceased altogether in about a week; and the disappearance of the pain permitted me to sanction a fish diet on September 30th. She did not, however, improve in her general condition; the anæmia (in spite of administration of iron) persisted, and she felt weak, the pulse remaining of low tension, but not rapid (70). Accordingly she was still kept in bed, and we hoped that the prolonged rest would be beneficial. On October 5th the evening temperature was 99·2, or higher than it had ever been on any of the seventeen days that she had been under observation; it was at the same level on the next morning, rising to 99·8 that evening, and to 101·6 on the morning of the 7th, and continuing to rise until it reached 102·6 on the evening of the 8th. (See Chart 1.)

Beyond flushing of the cheeks, and some coating of the tongue, the patient’s condition during this first access of pyrexia had not altered one whit. There was no sore throat; no return of pain or tenderness in the gastric region; the bowels were just as confined as before, but an enema which brought away some solid fæces caused no diminution in the pyrexia. She complained of a headache on the 11th, but this passed away in a day or two. She also occasionally perspired rather

freely at night. As the days passed, and the pyrexia increased rather than abated, she was repeatedly examined with care to detect any signs that could reveal its cause. The tongue became cleaner, and remained quite moist, although the temperature mounted up to 104° on the 19th, i.e., the fifteenth day of the access. On the 22nd, as I told you last time, the urine gave Ehrlich's diaro-reaction, and also showed an excess of indican. But there were no rose-spots on the skin; the abdomen was flattened rather than distended; the margin of the spleen, which a week previously could be felt below the ribs, was no longer palpable; the cheeks were still very flushed, and a *tâche* could readily be obtained by drawing the finger across the abdomen. There were a few scattered rhonchi over the lungs, but no cough. The cardiac sounds were feeble, and a soft systolic

endocarditis; but this could not be substantiated, for there had been no bruit on admission, and the pulse never rose above 100; nor were there any signs of embolism. We did, however, act as if the case were one of enteric fever, placing her upon liquid diet. A dose of antipyrin (5 grs.) with antifebrin (3 grs.) was given on the 24th, when the temperature reached 104.4° (it had three days previously been as high as 105° , when cold sponging was followed by a fall to 102°), which produced a fall to 101° . These antipyretics were repeated on two or three other occasions, notably on the 28th, 29th, and 30th, and exerted a far more powerful effect (see temp. chart) in reducing the temperature, but as on each occasion the fall in temperature was accompanied by shivering and depression, it was felt wiser to abandon these powerful remedies. For the most part she slept



murmur had become audible at the pulmonary cartilage.

Naturally, this persistent and continued pyrexia favoured a diagnosis of enteric fever; and seeing that she had been in the ward for eighteen days before it commenced, it was thought that if this were so, the fever must have been contracted in the hospital. But during the whole of this time the only other case of enteric fever in that ward was one who was well advanced in convalescence, and who occupied a bed at the very opposite end of the ward. The possible occurrence of local inflammation with suppuration connected with the presumed gastric ulcer seemed to be negatived not only by the entire absence of pain and tenderness, but also by the type of the fever itself; it was continued rather than remittent, nor were there any rigors. Then the appearance of a cardiac bruit suggested the possibility of ulcerative

well at night, but there were indications of increasing nervous prostration in slight tremor about the lips, and in mental depression. The pulse, too, began to flag, and some alcohol was prescribed. Still the tongue remained perfectly clean, and on the 28th, after some days' constipation, an enema brought away a copious solid motion. The case was now approaching to a solution of its mystery. There had been no change on October 31st (the twenty-seventh day of the febrile access), and as the temperature rose to 104.2° , a dose of 10 grs. of quinine was administered. No immediate effect followed. At midnight she began to shiver violently, the temperature fell to 100.4° . At the same time she complained of pain in the hypogastric region, and the bowels were opened thrice; this was followed by a rise of temperature to 104° . Quinine (10 grs.) was again given, but was vomited. Fomentations to the abdomen

relieved the pain somewhat; but when I saw her on the morning of November 1st, there was no doubt as to what had happened.

There had been no return of the vomiting; but she was pale and prostrate,—pulse 120. The abdomen was not distended, and there was some movement of its wall in respiration. But there was superficial tenderness over the right iliac and hypochondriac regions, and on percussion, a resonant note was clearly elicited all over the area of normal hepatic dulness. Four hours later Dr. Cayley saw her in consultation. The abdomen was distinctly fuller than in the morning, and tympanitic. The note in hepatic region was more markedly tympanitic. She had been sick on taking some milk; and the pulse had mounted to 132. The tongue remained moist and clean. Everything pointed to perforation, and the probability was that it was perforation from typhoid ulcer. We discussed the propriety of seeking surgical intervention, but in the patient's extremely feeble state we considered that laparotomy, even as a last chance of saving life, could not possibly succeed. There is little to add. She suffered no pain—the administration of opium removed that—but the tympanites increased, the pulse rose to 140, and she died at 9 p.m. on November 2nd. It is a singular fact—by no means, however, unknown in the history of enteric fever—that the mother of the girl and two other of her children died from the same fever some years ago. There is no doubt a family proclivity to certain specific fevers, and often to severe and fatal attacks.

The post-mortem examination by Dr. Voelcker revealed more extensive intestinal lesions than might have been anticipated from the clinical characters of the case. There was free gas in the peritoneal sac, and a quantity of liquid fæces with purulent exudation at the posterior part; there was some general peritonitis, "but," adds Dr. Voelcker, "this cannot be described as intense." The coils of small intestine were injected, but not distended; and about ten inches above the ilio-cæcal vulva a small perforation was visible in the wall of the gut, surrounded by necrotic tissue. The affected portion of the small intestine is before you, presenting a combination of recent infiltration of the lymphoid glands with ulcers in all stages, from small circular areas of pale medullary-like swelling surrounding a central slough, to larger

oval and irregular ulcers with undermined margins, and base formed of muscular and peritoneal coats, and some in which healing is already advancing. These lesions commenced about four feet above the valve, and I cannot do better than quote Dr. Voelcker's detailed description of them. At that level "there is," he says, "a slightly swollen and brown Peyer's patch. Three inches lower is a swollen Peyer's patch having a small early ulcer in it at each end. The next patch has an early ulcer with raised edges and a circular loss of substance at one side; four inches lower is a small discrete ulcer with an adherent slough; an inch lower a large Peyer's patch is swollen and shows a cribriform condition, with four or five distinct ulcerated small areas exposing the muscular coat; no slough. To one side is a swollen follicle with a yellow slough in it. Then follows a clean ulcer with undermined edges and no slough; then an ulcer almost perforated (torn on removal) with no slough; and then a very large pink swollen Peyer's patch extensively ulcerated, and still having small portions of slough adherent. It is here a perforation has occurred, and here peritonitis is most marked. In the last ten inches (of ileum) the Peyer's patches are both very swollen (recent infection) and also show old clean ulcers, as well as recent ones, the sloughs of which are not separated and only separating. The solitary glands are much swollen. The evidence is in favour of typhoid re-infection, there being present in the same portion of intestine two distinct stages of 'enteric' change. One small ulcer in cæcum; solitary glands in ascending colon much swollen, one ulcer in transverse colon, and two small ulcers in descending colon. These are typhoid ulcers." As regards the other organs, the spleen was soft and swollen, weighing eight ounces; the liver pale, fatty, and friable. The stomach mucous membrane was mammilated, especially towards the pyloric end; no ulceration; no scars, and very slight pigmentation of the mucous membrane. The kidneys showed cloudy swelling. The pericardium and pleura were natural; the heart wall pale, valves natural; lining membrane blood-stained, left ventricle slightly dilated; a few patches of collapse and hypostatic congestion of the lungs.

The value of a post-mortem examination in solving clinical doubts is here well exemplified.

In the first place, it proved that the diagnosis of gastric ulcer was unfounded. The condition of the gastric mucous membrane was at the most that of chronic catarrh, but such as is often unaccompanied by any symptoms during life. Secondly, the character of the intestinal lesions pointed to the case being one of so-called "relapsing typhoid," and compels us to the conclusion that when the patient entered the hospital she was already suffering from enteric fever. It is true we cannot fix the date of onset of her illness, but it is probable that her gastric disturbance marked its early course and dominated it. It was, indeed, a case of "gastric fever," which in most instances, I suspect, is really mild enteric. And the mildness of this primary attack was such as to dispel any suspicion of the real illness from which she was suffering. It ran a course which was practically apyrexial, so that the term "fever" is almost a misnomer in the case. The relative paucity of advanced and healing ulcers in the intestine accords with this view, which also accounts for her being able to continue her duties up to admission, whilst at the same time the persistence of her debility after the gastric symptoms had passed is explained now that we seem to have a clue to her actual state. The "relapse" proved to be more severe than the primary attack, and was especially characterized by high and persistent pyrexia. Thirdly, it is remarkable that diarrhoea should have been conspicuous by its absence until the very moment of perforation, and that, further, there should have been practically no distension of the abdomen. Fourthly, the occurrence of perforation took place with unusual mildness; but there was no doubt as to its having taken place about midnight on October 31st to November 1st. This is explained by the almost pinhole size of the necrosed opening, which further accounts for the comparatively slight amount of general peritonitis. Lastly, the case is of interest from the absence of rose-spots, and the urine yielding Ehrlich's reaction.

Now, although it seems clear that the disease could not have been contracted in the hospital, it may be asked whether the non-recognition of the primary attack did in any way favour the relapse. The patient had been on a fish diet for seven days before the first signs of febrile disturbance, marking the onset of this re-infection, appeared. But such diet could hardly be charged with being the cause of this re-infection in its fresh and wide-

spread infiltration of Peyer's and solitary glands. Had it been harmful, it would have surely been seen in exciting ulceration, delaying healing, producing diarrhoea. So soon as the fever was declared she was again restricted to liquid diet; and that cannot be blamed for the fatal perforation. No; in this case, as in many others, the relapse proved far more severe than the primary attack, and had it not been for the relapse, the fact of her having had enteric at all might never have been known. But I cannot think that on any theory the relapse is to be ascribed to dietetic error.

One other point arises out of the experience of this case, and it is this. Should an operation have been performed to wash out the peritoneal cavity, excise the perforated ulcer or suture it? The minute size of the opening; the fact that the signs of perforation were detected within ten hours of its occurrence; the mildness of peritonitic symptoms, might surely be urged as points in favour of recourse to this desperate expedient. But when one looks at the actual state of the bowel itself, it is impossible to believe that any surgical measure could successfully cope with lesions so extensive and severe. The treatment of perforation of intestine in enteric fever by laparotomy has been attempted now several times, with almost uniform want of success. The conditions are not to be compared with those of a perforated gastric ulcer, either as regards the local mischief or as regards the general condition of the patient. Here in this case, perhaps the local conditions were as favourable as one may ever expect to find them in enteric fever; but the vitality of the patient reduced by three weeks' high and continued fever, the cardiac enfeeblement, are sufficient to more than justify our decision not to submit her to so serious an operation, which could only have ended in disaster. And this, it seems to me, will ever be the great deterrent to the adoption of this—the only resource in face of perforation in typhoid, and a resource doomed beforehand to fail. Perforation of the ileum by mere extension of the necrotic process incidental to this disease is an accident, against which it is right and wise to take all reasonable precaution in the way of diet, of rest and such prophylactic measures; but it is unavoidable, and is hardly to be foreseen and never to be prevented, as it is the natural outcome of the morbid process itself.

(To be continued.)

A CLINICAL LECTURE ON COMMON DISEASES OF THE RECTUM.

Delivered at University College Hospital, Nov. 18th, 1895,

By CHRISTOPHER HEATH, P.R.C.S.

GENTLEMEN,—I shall speak to-day about the common affections of the rectum. In the ward you have just seen a man on whom ten days ago I operated for piles, and this is one of the common affections of the rectum; but when you get into practice, you will meet with many cases of piles which do not require operation. For instance, a woman comes to you and says she has a feeling of weight about her rectum, and that she is never quite comfortable. She does not get the relief she should when her bowels act, and sometimes blood passes with the motion. You may find that she is a middle-aged woman, has probably had two or three children, and that these symptoms always become aggravated when she becomes pregnant. That is very likely a case of internal piles, and it is your duty to examine and ascertain whether it is so, and it is, of course, necessary for you to know the feel and aspect of a healthy rectum before attempting to diagnose a diseased one. With your finger in the bowel you will be able to feel that there is more or less enlargement of the hæmorrhoidal veins and hypertrophy of the mucous membrane around, forming prominences in the rectum of varying size. An operation may be contra-indicated by the existence of pregnancy; and let me warn you never, if you can possibly avoid it, to do an operation upon a pregnant woman, for, however slight the operation may be, you will very likely produce abortion, from which very serious results may accrue. If it be a case in which operation is contra-indicated, you must, of course, temporize, and treat the patient in other ways; so also with many slight cases where the necessity for operation does not arise. The first thing, then, is to see that the patient's liver is doing its work properly, for in people with congested livers we very commonly find internal piles. Again, persons who do not take proper exercise very often have large hæmorrhoidal veins, which bleed from time

to time, but in them the loss of a little blood is rather beneficial than otherwise.

First, then, regulate the patient's bowels, and get the liver to act. For this, a mild laxative is better than a powerful purgative, and there are several such laxatives; sulphur is a favourite one—a teaspoonful of milk of sulphur taken in a little milk every morning will give a healthy evacuation, and very much relieve the patient. If that is not strong enough, you may use some other preparation, such as the compound liquorice powder—a teaspoonful or two stirred up in water and taken every night or the first thing in the morning. Cascara is one of the fashionable drugs at the present day, and a teaspoonful or two of the elixir of cascara will produce a fair evacuation. The objection to it is its exceedingly disagreeable taste. In addition to such medicines, you can produce an evacuation, in people who are habitually costive, by injecting a little glycerine into the rectum. A few years ago this was a very fashionable proceeding, and all chemists sold little syringes for the purpose. A better plan is to use a suppository containing a drachm of glycerine, the suppository being allowed to melt in the bowel. But among medicaments for the rectum there is nothing better than cold water; in the first place it acts as a stimulant to the bowel, and in the second place it has a tendency to constrict the vessels, and helps to restore the circulation to its normal condition. Whatever the disadvantages of water-closets may be from a sanitary point of view, they certainly enable persons to wash themselves after evacuating, which is far preferable to the habit of using paper. The Eastern nations do wash after an evacuation, and in that respect are very much cleaner than we are, and there is no reason why Europeans should not wash themselves after that function, as a quantity of clean water can easily be put into the pan of the closet for the ablution. Where an injection is necessary, I always recommend the individual to carry a Higginson's syringe to the water-closet and throw up cold water, and there is no need for the complicated apparatus occasionally seen.

If the case is one in which there is some little continuous worry—I will not call it pain—it may be wise to use some form of sedative to the bowel. The old-fashioned gall and opium ointment is not at all a bad one, but is objectionable on account of

its faecal colour. Belladonna ointment has the same drawback as gall and opium, but one grain of sulphate of atropia mixed with 1 oz. of lard makes a very good ointment; it can be introduced on the finger, is soothing, and gives great relief.

Patients who have trouble about the rectum should, as a rule, evacuate their bowels at night. We healthy people go to the water-closet, as a matter of course, after breakfast; but sufferers from the rectum cannot do that. If they evacuate in the morning they are miserable for an hour or two afterwards; therefore it is important for their bowels to be relieved the last thing before going to bed. Then they have eight to ten hours in a horizontal position, and the bowel has time to re-adjust itself by the time of rising. This is particularly applicable to business men and those who have to leave home at a certain time every morning.

If the case is more severe than those I have



Fig. 1.

described, you may have reason to operate; and, in order to do so, it is well that the patient should get the piles down, to enable you to see them properly, and get at them thoroughly. Therefore the patient should first have a purge, followed by an injection, and should then strain to get down the piles before you operate. The accompanying

wood-cut, fig. 1, from the late Mr. Curling's work on Diseases of the Rectum, shows very well the appearance of internal hæmorrhoids, which have protruded from the anus, and I would particularly call your attention to the delicate skin of the anus, which is everted with the piles. This is not so highly coloured as the mucous membrane, which protrudes and forms the piles, which are usually more or less purple in colour.

In the case of the man in the ward, those who were with me at the operation will remember that the first thing I did was to dilate the sphincter. Let me beg you to do that in every case on which you operate; it is a comparatively modern improvement in operating for piles, but its value is very great. When the patient is thoroughly under chloroform, you put your fingers into the anus, and steadily but forcibly dilate the sphincter. The advantage is that, without actually tearing the sphincter, it is paralyzed for the time, and you are then enabled to draw the piles down; another advantage is, that the sphincter, being paralyzed for a day or two, the patient has none of that wearying, constant contraction of the anus which gives rise to so much pain, and which was always present in former years.

The piles being protruded, there are half-a-dozen operations which you may use, and the one I generally employ in well-marked case of piles is to apply a ligature. The modern operation of applying a ligature is very different from the older one, when it was the custom to transfix the pile by a needle and tie it in two portions. The danger was that the needle which transfixed the pile very probably went through a vein also, and then when the vein was pulled wide open there was possibly suppuration, and the patient might get pyæmia, from which patients used to die; whereas such a thing is hardly ever heard of now-a-days in connection with piles. Draw down the piles with ring-forceps or a pile-hook, such as I show you, then take a pair of scissors and cut between the skin and the mucous membrane (fig. 1) so that you do not tie any skin in. That is very important, as the skin at the verge of the anus is very liberally supplied with nerves, and if you tie in a portion you will give excruciating pain to your patient for many hours. Having thoroughly isolated the pile, tie the ligature high up round the base. Then cut the pile off well beyond the ligature, and having dealt similarly

with the other piles, return the bowel with the ligatures, which hang out of the anus. But with the man who is in the ward I had something more to do, because he had external as well as internal piles; that is to say, the whole of the skin of the anus had become hypertrophied, and if I had left that, it would have been very bad for the patient. I therefore took two or three snips of skin away, cutting from the outside towards the anus, and then stitched together the edges of the cuts I had made. This stitching of the edges of the cuts is of great advantage to the patient, as the parts heal much more readily than when left to themselves, as they used to be, and I therefore strongly advise you to do this, notwithstanding that it makes the operation a little longer. Afterwards put a little iodoform into the rectum, and a suppository containing half a grain of morphia, and then lock up the patient's bowels for two or three days.

There are other methods of treatment, one being with the clamp. I do not like the clamp myself, but I show you one here. The clamp is not so well suited to internal piles, but it may be advantageously employed where the patient has got a quantity of loose mucous membrane, which comes down at every evacuation and protrudes, although there may be no distinct piles. In those cases you can clamp up one or more portions, and, having screwed the instrument up tight, you can then cut the mucous membrane off close, and sear the surface thoroughly with a Paquelin's cautery.

There is one form of external pile to which I will direct your special attention, because you are much more likely to encounter it in private practice than in hospital. It is a small matter of only inconvenience, and the lower classes will often stand a good deal of inconvenience before applying for relief. A patient will call upon you and say that he has been dining out a little more than usual, or his bowels have been constipated, and he finds he has got a little lump by the side of his anus. Never prescribe for any anal affection without examining; you must see for yourself, and ascertain what is the matter, and you will probably find a little bluish ball, which is very well represented in fig. 2. There is no inflammation, or very little, merely something which looks like a bluish pea, which is tender, and worries the patient when he sits down. That is nothing more

than a clot in one of the inferior hæmorrhoidal veins, and the proper treatment is to make the least incision into it and squeeze the clot out. This will immediately relieve the tension, and healing will at once ensue if a little simple dressing, such as cotton wool and iodoform, be applied. If that is not done, however, one of two things may happen; suppuration may take place around the clot, when a little abscess will form and discharge itself; or, a more frequent sequel is that the clot undergoes absorption, and a little loose fold of skin is left there. This is the explanation of the small folds so often seen about the anus.

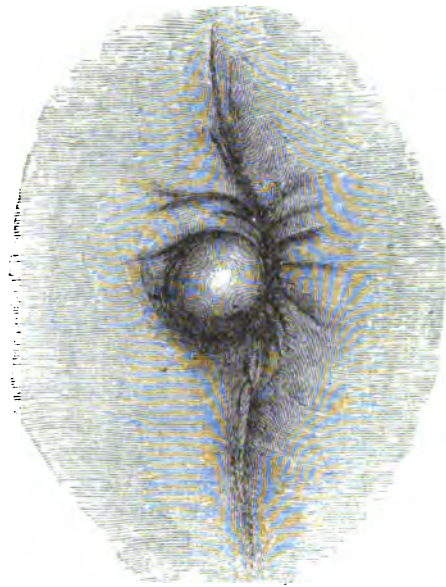


Fig. 2.

Now let me say a word about that extremely painful condition of the anus, which we will call fissure. I purposely have not said that piles are painful, because, as a rule, they are not so, but only wearying and troublesome. But when a patient comes to you complaining that when he evacuates he has a very sharp pain, lasting for perhaps an hour, so that he is compelled to lie down, you may safely infer that he suffers from fissure. You are bound to examine, and you can generally see the fissure if you get the patient to kneel up so that the light enters the anus, and then gently open it. You will find the anus considerably contracted, and the patient will not like the passing of your finger, but the finger should

first be smeared with vaseline, and should be introduced with a spiral motion, not a straight thrust. Down one side of the anus you will find a little crack in the mucous membrane, the explanation of which in most of these cases, at least in the male, I believe, is that the patient has had a very costive motion, and has had to pass through his anus a large mass of hard fæces, so that the parts become a little over-distended and possibly torn. In the female there is no doubt fissure does sometimes occur in labour, the woman has a bad labour, and the effect of the stretching of the parts for the passage of the child's head is to leave her with a fissure.

In the treatment of fissure, you may use palliative or radical measures. If the fissure has only just occurred, it will very often get well under the treatment I spoke of in connection with internal piles,—belladonna or atropia ointment, and keeping the bowels relaxed. But if the fissure occurred a week or two before the case is seen, that treatment is inadequate, and by far the best method in such cases is to dilate the sphincter. I know it is painful for the moment, but the relief subsequently afforded to the patient atones for the pain of dilatation, and in the majority of cases it is not necessary to administer an anæsthetic, though in a nervous woman this should be done.

But there are cases of extensive fissure, where it may be necessary to divide the sphincter partially with the knife, and for that you should lay the patient up and give him an anæsthetic. In an old-standing case, in which there is much induration, it may be better to pass a finger into the bowel, and guide upon that a bistoury, withdrawing the finger and knife together. The result is that the knife cuts into the fissure and divides a few fibres of the sphincter. It is never necessary to divide the whole sphincter; any one who does so really causes his patient a mischief, by paralyzing the bowel and causing him to lie up longer than is necessary.

Another troublesome affection of the anus is pruritus ani, and subjects of this suffer very considerably. The story is this:—A patient finds, on going to bed and getting warm, that he gets the most intolerable itching about the anus, and that this itching not merely keeps him awake, but almost necessitates rubbing the part. Continual rubbing will produce a condition of eczema such

as is illustrated in this drawing, though I have not myself seen one quite so bad as here represented. It is well to eliminate any possible cause for this irritation; and a very frequent cause in children is the presence of ascarides. But it is well to remember that thread-worms do occur in adults: therefore if you have any suspicion of their existence in the patient, you should take means to clear them out of the bowel, partly by purgatives and partly by injections. There is no better injection than the old-fashioned salt and water, which kills the worms, but their habitat is the cæcum, and purgatives enable you to remove the masses of eggs from which they are developed. Having done this, it may be well to make an examination of the rectum to see if there is a pile, which may keep up the irritation; or you may be obliged to fall back upon that refuge for the destitute—gouty diathesis, which has a good deal to answer for. The number of local remedies which have been applied to cure this anal irritation is wonderful. A 5 per cent. solution of cocaine painted upon the anus, after careful washing of the part, will sometimes give relief. Atropia I have also found very useful—one or two grains in an ounce of spermaceti ointment or lard. A suppository of 15 grains of cocoa butter put into the rectum and allowed to melt sometimes does good. You may, of course, apply opium or belladonna as well, but the mere presence of cocoa butter seems to give great relief. They are very troublesome cases, and the worst point is that after using each fresh remedy, which appeared at first to give relief, the patient returns, stating that he is as bad as ever; that he can get no sleep at night, and is miserable. It is no doubt a neurosis, and you must attend to the general health, eliminate every possible source of irritation, and then build the patient up in good health in every possible way.

Of course, the practitioner should always be on his guard as regards syphilitic manifestations in the form of mucous tubercle about the anus, i.e. large flat plaques on each side of the anus. When the patient stands up the two surfaces come into apposition; and I wish particularly to insist on that, because there is no question that a mucous tubercle will inoculate the surface against which it is pressed. Separate the surfaces, and then apply a folded piece of linen on which some white precipitate ointment has been smeared, and tuck

that in carefully between the buttocks. The affection is seen in syphilitic children, and is common in the lower class of prostitutes, but may occur in an innocent woman who has contracted the disease from her husband. Any form of mercury will do good. Some people prefer powder rather than ointment, and then the part should be thoroughly washed with soap and water, and dusted with calomel, afterwards a piece of fine lint or linen being placed between the affected parts. You must, of course, recognize that mucous tubercle is an evidence of constitutional syphilis, therefore the appropriate treatment of syphilis must be undertaken as well. Bear in mind also that mucous tubercles are communicable, and that a woman may convey the disease to her husband, and *vice versa*. Of course, one does not believe half the stories that one hears about water-closets, but it is a fact that women do sometimes pick up syphilitic disease on the dirty water-closets of railway stations and places of that kind.

The next common complaint I will speak about is the occurrence of bleeding from the rectum in children. A mother tells you that she notices a little blood in the child's motion, and that each time it evacuates the bowel comes down. It may be a case of prolapsus, but it may also be a case of polypus of the rectum. This latter is not at all uncommon in children, and with the finger you can at once ascertain whether there is a body hanging down by a narrow pedicle some inch or so within the rectum; if so, it is a polypus, and you should immediately, with your nail, tear the pedicle and take the polypus away. Though the polypus has been the cause of hæmorrhage in the rectum, sometimes for months together, it is very remarkable that you can tear through the pedicle and have no hæmorrhage at all.

But it may be a bona-fide case of prolapsus. This is not so very uncommon in children, and may be simply a result of debility; or it may be a symptom of more serious disease, such as stone in the bladder; but is more often a case of debility. A badly nourished child is put on one of those wretched chairs with a hole in the bottom, in order that it may evacuate its bowels, and is often left there for an hour at a time, until it suits the convenience of somebody to take it out. During that time it continues to strain, and at last brings down the bowel. That is the explanation of the much

greater frequency of prolapse among children of the poorer class; the better classes take much more trouble with their children, and do not allow them to get into those bad habits.

The way to cure a simple prolapse is to prevent it from occurring for a few days, and if the mother carefully carries out, for a week, the following instructions, the trouble will generally end. Show the mother how to put her finger upon the verge of the anus, and draw it to one side, and so convert the circular anus into a slit; then the bowel is puzzled and cannot come down. I have done that over and over again with success, and matters are expedited by giving iron and attending to the feeding. I am not going to speak of the more serious cases of prolapse to-day.

The last common anal affection to which I desire to allude in this lecture is ischio-rectal abscess, with its resulting fistula. From time to time you will see cases of abscess at the side of the anus, which are generally due to some perforation of the bowel by a fishbone or pin, or by a small ulcer, through which fæcal matter has escaped, and set up an abscess. This is exceedingly painful, and almost prevents the patient from walking. The great point is to open such an abscess freely, after giving an anæsthetic, and see if it has already opened into the bowel. If it has, time will be saved by laying open the bowel, and if this be not done, a fistula will certainly result. In many cases the bowel does not require to be laid open, but if I find the abscess goes some little distance up the side of the rectum, I feel sure it will eventually burst through, and it saves time to lay the whole thing open at once. Many such cases are treated domestically, and then they degenerate into fistula.

Fistula in ano is divisible into complete, and incomplete; and I think the majority are complete, but the surgeon does not always know where to find the internal opening. There are comparatively few examples of incomplete fistula, and they are divided into two classes—blind internal, and blind external. These terms are used somewhat arbitrarily, for a blind external fistula is a fistula which opens outside and is blind internally; a blind internal fistula is an internal fistula opening into the bowel, but having no opening outside. In the great majority of cases there is both an external and an internal opening. The commonest

places for the internal opening of fistula are either between the two sphincters or immediately above the internal sphincter, though the fistula may occasionally run higher up. In the slight cases, when the internal opening is once found, there is no difficulty about the operation. All you have to do is to slip a probe-pointed director into the external opening, and insinuate it along the fistula to the internal opening; then, guided by your finger introduced into the anus, bring the point out through the anus, and run a sharp-pointed bistoury along the groove to divide all the tissues. That is far better than the old plan, which was this. The surgeon took a blunt-pointed bistoury and insinuated it up the fistula, then met it with the finger at the internal opening and withdrew them together, cutting through all the tissues. But what I have described does not complete the operation in many cases, because some of these fistulae are extremely chronic, and they branch in more than one direction, and you will not cure your patient by laying open only one branch. Having laid open the several fistulae, it is a good plan, with sharp scissors, to remove the overhanging skin, because these edges may fall together and practically close the fistula. The removal of this skin permits the fistula to granulate up from the bottom. Further, the modern treatment is much more humane than the old plan, for it used to be the practice to poke something into the fistula every day, which gave an infinity of pain. I never have a fistula dressed at all. I thoroughly lay it open, trim it up, rub a little iodoform along it, and then lay in it a little cotton wool. I do not plug up the rectum, but put a suppository into it and let the patient go to bed. In a day or two he has his bowels relieved, and that brings away the small piece of cotton in the fistula, after which the dresser or nurse syringes out the fistula with a little red lotion twice a day, and the patient experiences no pain. In fact the operation for fistula is now a comparatively simple matter.

Parotitis.—Isolate the patient, and let him rest in bed, taking light nourishment,—milk, soup, and eggs. Apply the following over the swollen parts: *Hyoscyamus oil*, *chamomile oil*, each 1 fluid ounce; *Sydenham's laudanum*, $1\frac{1}{4}$ fluid drachms. Cover with a layer of cotton. Keep the bowels regular and give three teaspoonfuls daily of the following potion: *Sodium salicylate*, $1\frac{1}{4}$ drachms; *sodium benzoate*, $2\frac{1}{2}$ drachms; *julep of orange-flowers*, 1 fluid ounce; *linden-water*, 4 fluid ounces. Rinse the mouth with the following antiseptic solution: *Thymol*, 4 grains; *carbolic acid*, $15\frac{1}{2}$ grains; *tincture of eucalyptus*, $2\frac{1}{2}$ fluid drachms; *water*, 1 quart. Also use it for irrigation of the ear.—(*Tribune Médicale*.)

CLINICAL LECTURE

INTRODUCTORY.

THE NATURE OF DISEASE.

Delivered at the Westminster Hospital, October 9th, 1895,

By W. H. ALLCHIN, M.D., F.R.C.P.,

Senior Physician to the Hospital.

GENTLEMEN,—The formal Clinical Lecture, apart from the actual bedside-teaching and the lectures of the systematic course, appears to me to offer a convenient occasion for the consideration of a variety of subjects which find no suitable opportunity for discussion at our other meetings. Such for instance are individual symptoms common to several very different diseases, e.g. "pain in the back," "head-ache," "breathlessness," and the like, or some more distinctly objective condition such as "tympanites"—signs and symptoms, that is, which coming under notice in connection with individual cases in the wards, nevertheless require a more comprehensive consideration in respect to the very varied diseases with which they may be associated. But over and above such distinctly "clinical items" as these, there remain also certain questions of a more general and I admit of a more theoretical character which require some justification for their inclusion among the subjects of a clinical lecture, and such an one as I propose to speak of to you to-day falls into this category. For "clinical medicine," entailing as it does special modes of investigation and trained methods of observation, is essentially practical. "It is that department of medical knowledge which occupies itself with the study of individual cases of disease, as distinguished from that other department of medicine which occupies itself with the study of disease in general. It is that part of medicine which guides you to the investigation and discovery of the symptoms and signs of disease, to the classification of their relations, to the interpretation of their meaning, and to the elucidation of their bearing upon the general system, and upon the course of the disease of which they are indications. It is that part of medicine which enables you to determine the nature of the particular disease with which you are dealing, to predict its course, to forecast its issues, and to determine the means most suitable for averting death, relieving suffering, or bringing about a cure. . . . It is for

the sake of clinical medicine that all your previous studies have been undertaken. It is for the sake of becoming practitioners that you have studied physics, chemistry, anatomy, physiology, therapeutics, and pathology. For all these branches of knowledge supply information absolutely essential to the accurate and adequate conduct of clinical inquiry. . . . Clinical medicine is then at once the object and end of all your previous studies; it is in a sense the closing and the crowning of all your former work, and it is the means whereby you are to make yourselves sound and true practitioners in the art and craft of medicine." *

This being the nature and scope of clinical medicine, such a subject as the "Nature of Disease" requires, as I say, some justification for its consideration on an occasion like the present; and my plea is this. The work of the student consists for the most part of obtaining a knowledge of as many as possible of the innumerable facts and more or less precise observations which constitute the many branches of science going to make up the one denominated Pathology. Many of these data he is able to verify and observe for himself, but for a greater number he is dependent upon the statements of others which he learns at his lecture or from his book. Time alone is insufficient to furnish a knowledge of all that has to be known from personal investigation. Experience has convinced me that in face of the enormous extent of medical and allied science, the greater number of students are placed at a very considerable disadvantage, both in acquiring the needful information and being subsequently able to make proper use of it when acquired, for want of some connecting threads, some co-ordinating influences, some general ideas which may serve to give coherence and order to the seemingly chaotic mass of details with which they are faced. I am fully aware of the risks attendant upon making too much of general principles, which are most frequently of a theoretical character, and the special danger that they may come to occupy the mind, to the exclusion of more positive knowledge. But I feel equally sure that, for the want of some such guides, many facts are never completely understood, or the worker is left in hopeless confusion as to the real bearings of much that he learns. For exceptional minds, I

admit, such generalizations may be a hindrance, perhaps even positively harmful; but not so, I believe, to the majority, care being taken to emphasize the provisional nature of the principles enumerated, and to show that they are subject to modification with increasing knowledge, and only represent the point of view in harmony with the existing state of ascertained fact. A further and not unimportant advantage which I believe to be attached to these theoretical considerations is the help they afford to the recognition of the limits of what is known, and to formulate our ignorance. To this end, therefore, I propose to submit to you a "working idea" of what is meant by disease, as a convenient guide to you in the investigation of any individual case of illness.

One of the primary dicta of biology—of which pathology is a subdivision—is that every living thing may be regarded from the twofold aspects of "what it is," that is to say the thing itself, its structure and composition, and "what it does," or the energy it manifests. If to these be added the consideration of the surrounding circumstances—the environment—under which the living thing *exists* and *acts*, we have the entire range of possible directions of investigation, and to one or other of which heads every question concerning the subject of the inquiry may be referred, and this, it may be incidentally remarked, is equally true of inanimate objects. It is impossible to separate these fundamental conceptions. We only know the one through the other; we only recognize the material or structure by the properties and energies which it manifests, and conversely the properties and energies are inconceivable apart from the material through which they are rendered sensible. But—metaphysical speculation notwithstanding—it is practically convenient and possible to consider the phenomena of structure and function separately, at least to begin with, especially since each may be submitted to experimental observation.

Another generalization which lies at the foundation of our present knowledge, is that all phenomena appreciable to our senses, manifested by living equally with non-living bodies, are referable "to a common measure," and we refuse "to regard any vital process as understood unless it can be brought into relation with physical standards."* Among these physical

* Clinical Lectures by the late Sir A. Clark, Bart., *Lancet*, January 2nd, 1892.

* Prof Burdon Sanderson. Address at British Association for the Advancement of Science, 1881.

standards, the fundamental conceptions of the cosmos are the following:—(i.) That matter and force are each indestructible and are limited in amount. (ii.) That each is capable of undergoing alteration, the former in condition, the latter in direction; and that changes in condition of matter are associated with manifestations of energy, and reciprocally manifestations of energy imply some alteration in the condition of matter. (iii.) That matter is assumed ultimately to consist of indivisible particles called "atoms," which are for the most part grouped into molecules. And that such molecules are ever in motion, vibrating in varying planes; the various forms of energy being expressions of the modes of motion. (iv.) That the various forms of energy known as heat, light, electricity, etc., are intimately connected and correlated, differing according to the mode of motion exhibited by the constituent molecules of the material through which the energy is manifested.

Let us proceed now in the light of these ideas to consider what is really meant by structure and function, what ideas these terms precisely denote. The first and most obvious notion connected with structure is comprised in the subject-matter of gross anatomy which deals with the size, shape, position, relations and naked eye appearance of the various organs and parts which together make up the body. By means of the microscope the investigation may be pushed a great deal further, and the minute anatomy of the tissues of which the organs are constructed may be ascertained. Between these two branches of inquiry, which deal with the body as it is quite apart from what it does, no hard and fast line of separation exists; the one merges into the other, and is but a step beyond in the same direction. But structure does not stop here. Whilst no doubt there is a limit to the investigation of *visible* structure, a point, that is, beyond which the microscope however perfect cannot go, that limit is not yet reached. But even if it were, that would not be the limit of actual structure. There is a still further stage in the inquiry into what a living body, or any part of such a body, really is, and this is known as the "chemical composition" of the material, and further its ultimate "molecular constitution," and when that is reached, the structure of an object in its fullest and proper

sense may be regarded as known. This last stage is still within the realm of theory, but the hypotheses that are current are strictly in harmony with the fundamental conceptions upon which all our knowledge of the material world is built. Of the chemical composition of the tissues much is known, but it promises to be the most fruitful field of discovery. Chemists have for many years past been compelled to extend their ideas of the composition of substances, and they now speak of "chemical structure," meaning thereby not only the actual chemical elements and proportion thereof of which a substance is composed, but the arrangement of the atoms of these elements in the molecules and the relations they bear to one another, and intimately dependent upon which are the properties of the substance. Chemical structure, then, may be taken as the third and final term of the series of which gross anatomy and general or minute anatomy are respectively the first and second, and as no distinct line can be drawn between the latter, so for my part do I conceive of no absolute distinction between the second and third. In regard, however, to the structure of the body, it is not sufficient merely to consider it at any one time in the course of its existence, but its nature and appearances from its origin during the successive stages of growth and development until maturity is reached, and the subsequent changes which take place during decline to death are also to be regarded. A knowledge of the anatomy of the adult animal or plant, however accurate, still falls very far short of completeness, unless it be considered in connection with the progressive phases of development through which it has passed, and to this extended conception of anatomy the term morphology is applied.

Inseparable from the idea of matter, the structure of which has been investigated, are the properties by which it is recognized. Such are weight, hardness or softness, flexibility or rigidity, elasticity, permeability, and the like, all dependent on the character and degree of the attraction and repulsion between the ultimate constituent molecules of the material. These and others may be denominated physical properties, and are possessed by all matter, whether living or not. Certain substances, however, over and above these qualities, manifest certain specific properties, in virtue of which they are called living, and conformably

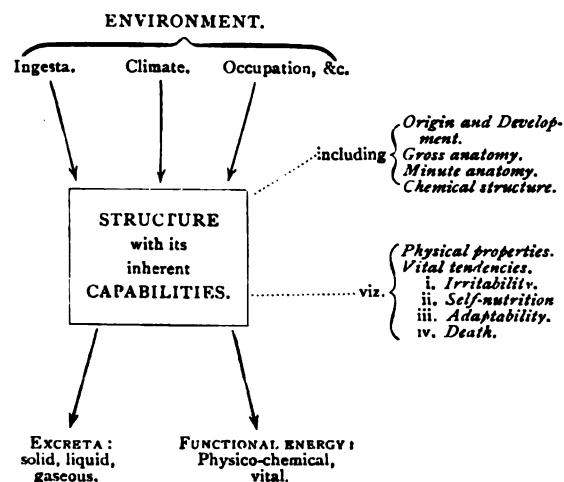
to the principle above laid down, we assume that these properties are the outcome of some special molecular structure. These peculiarly vital properties may be arranged in four groups. Of these the first are those which may be collectively termed "irritability," that is, various forms of response to the application of stimuli (for the most part conditions of the environment) which we recognize as muscular contraction, nervous energy, and glandular secretion, together with the power of multiplication and perpetuation of the race by fission, gemmation, etc. The second group of characteristic vital properties are those comprehended in the processes—largely chemical—by which the living material increases in bulk and maintains its structural integrity by the conversion of outside matter—food—into its own substance; and these may be expressed as "trophic" properties, or tissue metabolism. Within a very variable range, living matter has, as one of its most significant peculiarities, the power of adapting itself to variations of the external conditions subject to which it exists. Within certain limits of temperature, barometric pressure, quality of food, etc., living things may adjust themselves, beyond which they succumb. This is sometimes expressed as the resisting power of the tissues, one highly-specialized phase of which is known as "immunity." Lastly, all living things, sooner or later, cease to display their vital characteristics, and die. Concerning each of these groups much might be said, did time permit, in a general way; in detail, they form the subject matter of physiology. It is, however, desirable to remember that all the properties which may be included within these four groups are possessed by all living matter, though in a variable degree—certain parts being frequently specialized for the more complete and perfect performances of some of them. Moreover, they are not always equally manifest, but remain in abeyance until called forth; hence they may be conveniently designated "tendencies," in contrast to the *constantly* present physical properties. When these tendencies assert themselves and become manifest, they constitute the functions of the organism.

In addition to structure and function there is the environment, apart from which we cannot well conceive of material objects. Among the most

important external circumstances which influence the living body are; those comprised within the term climatic, viz. temperature, barometric pressure, atmospheric humidity or dryness, electrical states; ii., locality of habitation as regards soil, elevation, etc.; iii., the nature and character of the ingesta, solid liquid and gaseous, including therein such toxic bodies of whatever kind which obtain entrance into the body; iv., occupation. Of any of these it is impossible to lay down a fixed standard which alone may be regarded as normal. Each and all vary within wide limits of compatibility with a normal existence, but beyond these limits they become the determinants of disease or even death.

Given then structure, with all that is therein implied, possessed of certain inherent properties and tendencies, and subject to an environment of considerable variability, we have as an outcome certain physico-chemical and vital processes or functions which constitute the physiological life of the individual, together with material excreta, solid, liquid and gaseous. The original structure undergoes change and alteration in condition in virtue of the energy manifested, but is as constantly reconstituted in accordance with its inherent capability of self-repair. In other words, the living organism duly supplied by food and under the influence of satisfactory surroundings effects a transformation or redistribution of energy with a coincident change in material.

These ideas, to which of course I make no



claim for originality, I have been accustomed to express in a semi-diagrammatic form, with the

advantage, as I believe, of rendering them more clearly intelligible in their relations to one another.

Observation and experience has taught us to recognize a certain range of structure, of function and of environment as being associated with what we agree to regard as healthy life, departures from which we equally consider as disease. The notion of disease implies no new element, but simply some perversion of the factors that together make up health. Disease therefore, as has been well said, is the "correlative of health." And since the standard of health cannot be defined with precision, so no absolute hard and fast line can be drawn between these two states of being. It is clear, therefore, that there is not one single standard of health, but on the contrary there are several types of health. I myself have been in the habit of using the word "temperament" to designate these several ways of being well, though I am fully aware that the term has been employed in other senses. Health and disease are both objective and subjective in their relations; each is a condition recognizable by the observation of an outsider, and each is a state appreciable only to the possessor. Disease usually, but not invariably, implies discomfort, dis-ease, or even pain, though this is a vague criterion, and a fatal malady may be associated with scarce a symptom complained of by the patient.

It would necessarily follow from such a view of the nature of disease, that all disease is both structural (organic) and functional. Both conceptions are equally involved in what we mean by the expression. There can be no structural abnormality however minute, without some functional perversion; and conversely a functional imperfection necessarily implies a structural deficiency. But just as there are many functional manifestations both of health and disease which are not accompanied by any structural change recognizable by the means at present at our command, so many structural abnormalities may be found post-mortem without any functional defect obviously connected therewith.

Such a way of considering the subject has the further advantage of enabling us to appreciate the direction in which the causes of disease produce their effects. Whilst on the one hand inherent imperfections in the structure or its properties or tendencies account for so-called

hereditary maladies, those which are acquired find their determination in some fault in the environment; or, as seems probable in a large number of cases, the actual disease is the resultant of a combination of both extrinsic and intrinsic shortcomings.

The scheme also furnishes a basis for the classification of the various more or less distinctly separate diseases which we are in the habit of recognizing. From the structural point of view they may be grouped according to the organ or region affected, or to the tissue which is involved, though beyond that, at present, we can hardly go. Whilst from the functional side four main groups of morbid processes are readily distinguishable, corresponding to the four main tendencies and functional manifestations which all living matter exhibits. Thus a very large number of diseases to which special names are attached essentially consist, in their functional aspect, of defects of nervous or muscular, or glandular irritability, whether in the direction of excess or deficiency or perversions. Another great group of maladies are fundamentally imperfections of the metabolic processes constituting tissue nutrition, whether on the constructive side that is connected with the due elaboration of the ingesta, or in respect to the subsequent tissue destruction and consequent waste formation and elimination. A weakened resisting power on the part of some one tissue or organ, or of the entire body, characterizes a third group of infirmities. And a proneness to degeneration and premature senility or death is the leading feature at the root of others. There are very few of the ills to which flesh is heir which do not find a place somewhere in this category, even the specific infective diseases which are the furthest removed from normal physiological processes may be accounted for.

Without doubt, from the side of clinical medicine, it is the functional (dynamic) aspect of disease with which we are mainly concerned, although along with it the structural (static) condition is the first object of investigation, and even may be ultimately of treatment. By the method of "physical diagnosis," the evidence derivable from both sources is ascertained; but for the complete comprehension of a case the "vital diagnosis" is all-important, and its subject-matter specially concerns the functional manifestations of the patient, conditioned by such circumstances as age and sex.

By this latter is the personal equation of the individual made known, the peculiarities of his or her special vitality disclosed, and those idiosyncrasies of living realized which serve to distinguish the individual from the race.

This, then, I submit to you as a working idea of the nature of disease which will serve to co-ordinate and give coherence and order to the multitudinous phenomena which you will be called upon to recognize. It makes no claim to finality, but it is strictly in harmony with the principles and ideas which regulate our investigations of the material world. It is open to modifications and alterations as fresh laws and hypotheses become formulated, but meanwhile will serve to correct the vague habits of thought and verbal expression so easily acquired and so harmful in effect.

CLINICAL NOTES.

WITH DR. JOHN PHILLIPS IN THE GYNECOLOGICAL
DEPARTMENT OF KING'S COLLEGE HOSPITAL.

After-History of Three Cases of Total Extirpation for Cancer.

Case I.—This patient first attended here nearly five years ago, with an epitheliomatous mass occupying the posterior lip of the cervix. As the uterus was quite mobile, and the vaginal mucous membrane uninvolved, radical operation was clearly indicated. Total extirpation of the uterus and ovaries was therefore carried out, from which she made an easy recovery. She has attended here at regular intervals since the operation; in February, 1894, she was quite well and the cicatrix healthy. To-day the patient reports herself for the last time; she has had no hæmorrhage, pain or discharge, her weight keeps about the same, and her general health is good.

On examining her per vaginam we find the vaginal roof is a *cul-de-sac* as before, through which nothing definite can be felt. By the speculum the cicatrix is well marked, glistening,

but slightly shorter than a year ago; there is no bleeding or pain on free handling.

The patient has passed through the menopause. We may now consider her in all probability as cured.

Case II.—This patient aged 33, married and a multipara, came here two and a half years ago, with the complaint that for three months she always had considerable bloody discharge after coition. On examination a small growth was found sprouting from the anterior lip of the cervix, which bled very freely on being touched. The microscope proved it to be an epithelioma.

The patient was admitted and the uterus extirpated per vaginam, the ovaries being left. She made an easy recovery, and has since reported herself at three-monthly intervals; so far there has been no indication of a recurrence. To-day she says she has had no backache or hæmorrhage; when each monthly epoch comes round, she has all the sensations of menstruation, such as weight in the pelvis, nervousness and headache, but no flow.

Per Vaginam.—The condition is very similar to that observed in Case I. The cicatrix can be felt, soft and smooth; there is no bleeding on withdrawal of the finger. By the speculum, the cicatrix is whitish in colour, with healthy pinkish-red surrounding vaginal mucous membrane.

We can therefore conclude that, so far, this patient is also free from recurrence. This symptom of bleeding after coitus is a very important one, and may often be an early indication of the onset of serious disease. The patient must as a rule be questioned as to its occurrence, as from a natural modesty she will be reticent in alluding to it.

Case III.—This poor woman, aged 40, and a multipara, whom I told to come here to-day, first applied for relief eight months ago. She had then suffered for four months from continuous bleeding and much backache; there was no loss of flesh. We found on vaginal examination that both cervical lips were enlarged and the mucous membrane breaking down in places. There was much hæmorrhage after examination, necessitating her admission into the wards. The growth proved

to be glandular cancer, and being apparently limited to the uterus, total extirpation was performed; the ovaries were also removed. She made a rapid, immediate recovery. The patient visited this out-patient room in three months' time: the vaginal wound was not then quite healed, but it was soft and apparently healthy. Two months ago she again reported herself. Slight blood-stained discharge then followed vaginal examination, the scar was harder to the touch and the wound not quite healed, the finger could be pushed easily into a deep cone-shaped cavity. She complained bitterly of her back (sacrum). A return of the disease was evident.

To-day you hear she has had a recurrence of her metrorrhagia, her sacralgia is worse, and keeps her awake at night. She eats well and is not losing flesh. On vaginal examination, you can feel the two flaps of the former wound, hardened, infiltrated, and gaping, allowing the finger to pass up to the peritoneum; a certain amount of bleeding follows and the discharge is not sweet. You notice also her careworn expression.

We have here had three cases of the same disease and operation in review. One may be said to be cured, another remains well, the third has a recurrence. It is difficult to say why this last result has followed; probably owing to the woman's age being more advanced, to the delay in applying for assistance, and to the fact that the type of cancer was glandular, and not epitheliomatous, as in the first two. In all three the chief symptom was hæmorrhage, the operation total extirpation, and the immediate result good.

Inflamed Fibro-myoma Uteri with Cardiac Disease.—This patient, aged 40, and a multipara, you may remember applied here first six weeks ago. She then had a temperature of 101° and looked very ill. The abdomen was tender, and a hard fixed swelling could be made out in the lower hypogastrium. Vaginal examination showed complete fixation of the uterus. She was admitted into the wards for presumed perimetritis, and has been discharged a week. The patient complains still of much left-sided pain, with distress of breathing on exertion. On examination we find a hard, nodular, somewhat painful, centrally situated swelling, reaching to midway between the umbilicus

and symphysis; it is not fixed. Vaginal examination shows us that the uterus forms part of this swelling, which bimanually is mobile. The tumour probably consists of a number of subserous fibromyomata. With the history of recent perimetritis, it is inadvisable to pass the sound.

On examining the patient's heart, we find a loud apical systolic murmur, conducted into the axilla; this condition may probably cause her dyspnoea.

Here is obviously a case in which operation is strongly contra-indicated. As she can rest up at home, we will advise her to do so, and come and report herself from time to time. In the meanwhile the bowels must be regulated and an iron and arsenic tonic given. The fibroid requires no treatment at present, as it gives rise to no hæmorrhage, and, so far as we can judge, is not increasing in size.

Melancholia after Labour.—This patient is a young primipara, who has just lost her six weeks old baby. Her friend who comes with her tells us that for some time before this event she was "peculiar," but since, she has several times threatened to do away with herself, although there has been no actual attempt to carry out the threat.

You observe the sad, depressed look of the patient, and her despondent attitude. Her hands and feet are cold and her tongue dirty.

The condition of the bowels it is difficult to ascertain with accuracy. She tells us she hears voices whispering to her and advising her to do wicked acts.

On vaginal examination, we find a perfectly normal state of things; local treatment is therefore not called for.

This patient should not, on any consideration, be left alone, and we must take her friend aside and explain the possibility of the patient suddenly attempting to commit suicide or even murder. The hearing of voices is always a bad symptom. Free purgation, with careful and, if necessary, compulsory feeding, must be carried out for a considerable time. She will in all probability recover from the attack, which may be termed puerperal insanity.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 4, 1895.

TWO CLINICAL LECTURES

ON

ENTERIC FEVER.

Delivered at the Middlesex Hospital, on October 26th, and November 15th, 1895.

By **SIDNEY COUPLAND, M.D., F.R.C.P.,**

Physician to the Hospital.

LECTURE II.—(*Continued.*)

IN striking contrast to the case I have just related is that of another patient in the same ward, whose illness was brief, running its course within fourteen days, of which only eight were marked by acute symptoms. The patient, J. C., was a girl of sixteen who was stated to have been attacked with a "sick headache" on October 25th, and, as we were informed on her admission, who had been quite well up to that date. It was not till after her death that we learned she had been ailing for about a week previously, although she was pursuing her usual avocations all the time. This fact made all the difference in arriving at a diagnosis, for when she was admitted on October 28th we thought that she had only been ill four days instead of eleven. She was delirious on the night of the 25th, and since the 26th had been unable to recognize her friends. On the 28th diarrhoea set in. She was a sparely nourished girl, in a neglected condition, her hair abounding in vermin, and her body covered with stains and marks of flea-bites. She lay in bed in a semi-comatose state, eyes half open, head retracted, but not recognizing anyone nor being capable of being roused, but she resisted passive movements. There was mild delirium, and she was constantly waving her arms, and now and then there was twitching of the angle of the mouth. The pupils were dilated and sluggish, and there was some discharge from each ear. Lips and teeth covered with sordes. Skin dry; temperature 104.8° ; pulse 120 and small. There were no signs of thoracic implication. The abdomen was not distended; there had been slight diarrhoea. During the night there was an attack of tonic spasm, the limbs becoming rigid, the head

more retracted, and face became cyanosed. There now occurred involuntary micturition and defæcation.

With the limited history before us, the diagnosis seemed to lie between typhus fever, pneumonia, and meningitis. But there was no eruption, either characteristic of typhus or enteric; no signs of pulmonary consolidation; whilst as regards meningitis, the headache and delirium followed by stupor, the head retraction, and the convulsive spasm were in favour of this explanation, to which the occurrence of otorrhœa also lent support. The possibility of tubercular meningitis was favoured by the fact that two of her mother's relatives, viz. mother and brother, had died of "consumption." An ophthalmoscopic examination, however, revealed no changes in the optic disc, and I think the disappearance of the headache and the absence of vomiting may also be placed against this view of the case. No urine could be obtained for examination, so that we had no opportunity of availing ourselves of Ehrlich's test. During the 29th and 30th she remained in much the same condition, except that the retraction of the head became more marked, and the limbs were frequently moved spasmodically. The pyrexia ranged between 103° and 105° . Death occurred in the evening of the 31st, consciousness having returned shortly prior to this, and the temperature rising to 105° .

Thus she had only been in hospital four days, and her illness on the fullest estimate had lasted fourteen days, although at the time we only thought it had lasted eight days.

At the post-mortem examination by Dr. Voelcker the absence of definite signs of meningitis was noted; but it was remarked that "the meninges over both hemispheres are markedly injected, and on the left side are also slightly dull and more sticky than on the right." There was no tubercle seen, and no lymph. Both middle ears contained granulation tissue, but the membranes were not ruptured. The lungs were congested hypostatically, and there were a few broncho-pneumonic areas in the right lower lobe. In the abdomen the intestines did not appear distended. The spleen weighed four ounces, and was but slightly swollen.

The intestines are thus described: "Throughout the small intestine Peyer's patches present a well-marked 'shorn-beard' appearance, but only in the lower sixteen inches is there any definite swelling or ulceration. In this area the highest patch shows the pale pink, discrete but not pitted, swellings. A little lower down an ulcer with still adherent slough, below which is an ulcer running transversely from which the slough has almost entirely separated, leaving a clean base of muscular coat. In the lowest patch there are several ulcers with still adherent sloughs. No perforation. No ulceration of the large intestines." The small intestines had liquid contents; but there were scybalous masses in the colon. The mesenteric glands were markedly swollen.

Now such appearances denoted that the typhoid process must have been in progress nearly three weeks. Dr. Voelcker thought they were consis-

"known the head so rigidly retracted, that deglutition and breathing were impeded."

Time will hardly allow me to do more than refer very briefly to other cases of enteric fever that are still under treatment in the wards. There is, for instance, the case of J. T., the man, 42 years of age, who was admitted with bronchitis and mild pyrexia, without any distinctive signs of the underlying cause of these conditions; and who after having been allowed up for nine days experienced a relapse of fever, a few days after his son who had had a similarly marked primary attack was also the subject of a relapse. Well, he has had a prolonged second attack, giving us some anxiety; not so much from the extent of diarrhoea and other intestinal symptoms, but from the state of asthenia to which he was reduced. For more than two weeks his tongue remained dry and brown. The pyrexia, as you will see from the chart

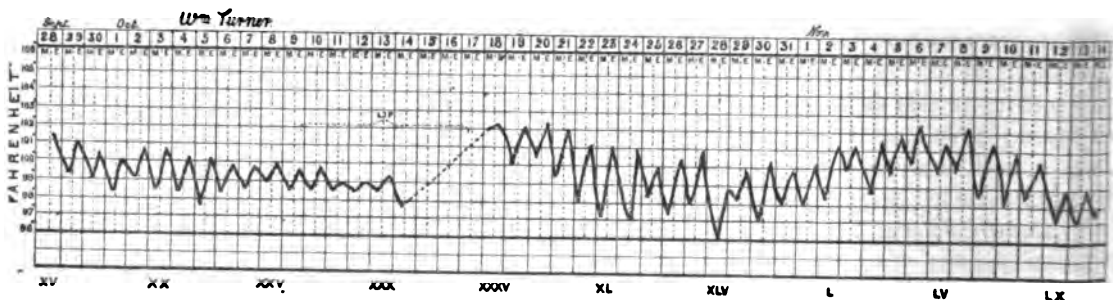


Chart No. 2.

tent with a duration of fifteen to eighteen days, and yet it was only for about a week that the patient had been really ill, so ill as to seek medical attendance. This affords another illustration of the insidious onset of enteric fever, whilst at the same time the case is exceptional in the violent onset of cerebral symptoms, and their marked predominance during the second week of an attack. They may be taken as an index of the severity of the systemic poisoning, and are far more likely to occur in typhus than in enteric fever. I can hardly recall another case of the latter affection in which there has been such marked retraction of the head and rigidity, and such continued coma as were here present; but Dr. Murchison says that "rigid contraction of the muscles of the trunk, neck or extremities is met with in some severe cases," and that "more than once" he had

(chart 2), has not been high, but has been persistent; and there has been no rash. The first typical "typhoid stool" that he passed was on October 21st, after an enema (there had previously been constipation), i.e. on the fifth day of the relapse; since then he has had one or two loose motions daily, the offensive character of which has been corrected by the administration of salol. The abdomen has never been distended, but the continuance of this relaxed state of the bowels—it could hardly be termed diarrhoea—led me to infer that there may be more lesion of the large intestine than usual, and to order a large enema containing tannin on November 3rd. This was twice given, and he expressed himself as relieved by it; but as there has been no further evidence in support of the view that the case is parallel to the one detailed in my last lecture, as he has moreover rallied from

his somewhat collapsed condition which might be ascribable to "faecal intoxication," the irrigation has not been repeated. I need hardly say that this is a procedure which must not be employed lightly, as it may do more harm by disturbing the intestines than good by cleansing the ulcerated surfaces.

We have two other cases under treatment, the one, J. M., a man æt. 36, admitted on October 30th, on the sixth day of his illness; the other, A. J., a woman, was admitted on November 5th, in the third week of her attack. The latter is the more severe case, there being much tympanites and a fair amount of diarrhoea, as well as marked pyrexia. I do not propose, however, to enter fully into the histories of these cases, but mention them mainly in order to show you the effect which "ice-cradling" has had upon the pyrexia in each case. In the case of J. M., the ice-cradle was applied first at 10 p.m. on November 1st, his temperature then being 103.4° . By 2 p.m. on the 2nd this had fallen to 101.2° , and the cradle was removed; but at 10 p.m., temperature 103.2° , it was re-applied and maintained continuously until 10 a.m. on the 5th, when the temperature was only 99.2° . (Chart 3) Since then the temperature

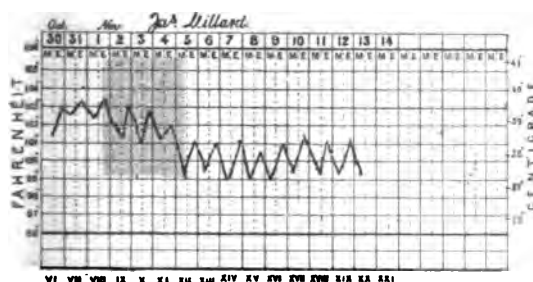


Chart No. 3.

has ranged between 99° and 101° , and the patient has in other respects improved; although he has complained of some pain in the right side, where a pleuritic rub can be detected. I do not think, however, that this pleurisy can be set down to his cold-air bath, as on admission he had a similar patch of pleurisy on the left side.

In the other case ice-cradling was commenced on the evening after admission, when her temperature was 104.4° , and was continued without intermission until the morning of November 10th, i.e. four and a half days,—when the temperature was 101° . (Chart 4.) It is premature to attempt to

draw any general conclusions as to this antipyretic method or to contrast it with cold bathing, from which we have hitherto had such good results; it

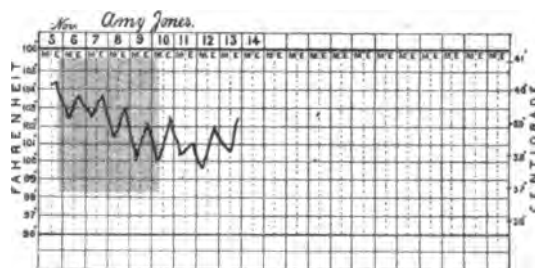


Chart No. 4.

does not produce so marked an effect as the bath, but the gain in its not involving such disturbance of the patient may counterbalance this.

A CLINICAL LECTURE

ON

GASTRIC PERFORATION.

Delivered at the Birmingham General Hospital, on
November 7th, 1895,

By GILBERT BARLING, M.B., F.R.C.S.,

Surgeon to the Hospital, and Professor of Surgery, Mason
College, Birmingham.

GENTLEMEN,—I have recently had under my care here an extremely interesting, although rather tragic, case of perforating ulcer of the stomach. The care of gastric ulcer is commonly allotted to the physician; the surgeon only sees such cases when the ulcer has so extended as to perforate through the whole of the stomach walls, and allow leakage of the contents of that viscus into the belly cavity. Therefore I shall not dwell upon the symptoms of gastric ulcer, as that does not come within my province, but I want to say something about the signs of perforation, and the subsequent treatment, which is allotted to the surgeon.

This accident is of considerable frequency; I think perhaps of greater frequency than is generally realized. The fact that I am particularly interested in the subject may have something to do with it, but I know of four cases of such perforation which have occurred during the last fortnight. One of them died on the way to the hospital

from shock. The girl on whose case I am lecturing to-day, was brought here and operated upon. The third case I saw in a town near here, but the girl was too ill to be operated upon; she was dying when I saw her. The fourth case was in Birmingham, and I did something for her relief. For one surgeon to see four cases in a fortnight shows that the condition cannot be very rare.

This perforation of gastric ulcer is a very fatal accident; I think it is one of the most fatal things which occurs. Then there is something about the cases which appeals to our sympathy very strongly. Most of the victims are young women, apparently in pretty good health except for indigestion, doing their work as usual, until they get the very sudden pain indicative of gastric perforation. I should say that fully 95 per cent of such cases die. Perhaps another very good reason for drawing attention to this condition is that it is not recognized as early as it should be, with the result that the proper line of treatment is not adopted, nor is the affection treated adequately when treatment is of most avail.

Before commencing my subject proper, I would like to remind you, by these diagrams, how the stomach really lies. Most of it is pushed up in the left hypochondrium; very little appears below the liver, and only a small portion of the stomach is readily accessible from any wound you can make in the abdomen. Whatever has to be done in stitching up the perforation generally has to be done inside the belly underneath the liver, with that heavy organ dropping down upon and very much inconveniencing the operator. The likeliest area of perforation is on the anterior surface near the lesser curvature, and nearer the cardiac than the pyloric orifice. From this drawing made from a frozen section, you will see that when perforation occurs towards the cardiac end, on the lesser curvature of the stomach, all this space underneath the diaphragm gets occupied by the extravasated food and bile and gastric juice. This is known as the left sub-phrenic space, and is a very common place for the gastric leakage to occupy. Above it is the diaphragm, below it is the left lobe of the liver and the stomach, to the left the spleen, and to the right is the falciform ligament of the liver. To make the matter quite clear, I also show you a drawing produced from a sagittal section, the original being in the

College museum. Notice how the stomach curves round the liver, and notice also the close relationship between the sub-phrenic space, the diaphragm, and the thorax. It is very important that you should realize this, because if the patient survives perforation of gastric ulcer for two or three days, an abscess is very likely to form here, and extending through the diaphragm, to set up empyema, pneumonia, and abscess of the lung. This process accounts for the death of many patients who struggle through the first few days after perforation.

Now to come to the case proper. The patient, aged 20, was a servant girl. It is in servant girls of about this age that we find most of the cases of gastric perforation. She was admitted on October 30th, at 6 p.m., and at the time was extremely ill, and collapsed to a degree. She complained of pain in her belly and vomiting. In her previous history she stated that she had suffered pain after food for several months, that it usually came on a short time after food, and that it was felt at the epigastrium and between the shoulder-blades. The patient had never actually vomited food after a meal, but had often felt sick, and at times had thrown up a clear, water-like fluid, but never remembers to have vomited blood: she had been pale for many months.

That, I should say, is a history of indigestion, but not a complete history of gastric ulcer, because in a typical history of the latter you would expect to get vomiting of blood. Still, I think it is a very important history in the light of what happened afterwards; therefore it should be kept in mind. The indigestion had been going on for many months, but until Monday, October 28th, she had gone on much as usual, and was able to do her work.

Now I come to the history of the perforation. On Monday afternoon, October 28th, as the patient was getting in some coal, she felt a very severe pain in the belly, between the umbilicus and the ensiform cartilage. When asked if she felt faint, she said she did not know about that, but she was so bad that she lost her sight. As you know, one of the early indications of fainting is generally that the patient cannot see for the time being; therefore it will not be exaggerating this patient's condition if I take it that she was faint. The pain was very intense, and made her feel sick. Shortly afterwards (not at the time of the intense pain) she did vomit.

Her bowels were not opened that day, nor were they between the time of perforation and the time of admission to the hospital, although she had an enema. After the intense pain and the faintness had passed off and the vomiting had come on, she noticed that the "pain went widely all over the abdomen." This diffusion of pain continued throughout the next two days, up to the time of admission. The abdomen swelled and became very hard, and the vomiting was frequent and severe. On admission she was very pale, her pulse 144 and extremely feeble; and her temperature 98°. The abdomen was extremely distended, tense, and very painful. On percussion it was found to be tympanitic in the central portion, and dull in the loins. Now comes a very important point: viz. liver dulness was absent, except for a narrow band in the fourth space, whereas, as you remember, it should occupy in front an area extending from about the fifth rib to the margin of the thorax. This meant that there was a quantity of free gas in the belly cavity, and that it had got in between the liver and diaphragm, obscuring the liver dulness. Of what importance was this? It told, in a very positive way, that there had been perforation, either of the stomach or intestine, and that gas had escaped into the peritoneal cavity. On palpation of the abdomen no special resistance could be made out at any particular point; it was all extremely tense together. Examination by the rectum and by the vagina gave no further information. The lungs and heart were normal. By the administration of brandy per rectum, and by hypodermic injections of strychnine, the condition of collapse was distinctly improved.

We have now come to a point at which I think we must try and sum up a little:—

Diagnosis. Here is a young woman, evidently anæmic, 20 years of age; by occupation a servant girl, with a history of indigestion. During exertion she suddenly gets a pain in the region in which the stomach lies. This pain is intense, and she is collapsed. During the next two days the symptoms of advancing peritonitis come on; there is pain all over the belly, distension, rigidity, vomiting. What could it be except perforation of gastric ulcer? One of the things we must always bear in mind in young people is the possibility of a perforated vermiform appendix; but the history I have been relating is not that of acute appendicitis. Acute appendicitis begins with a general colic

pain about the umbilicus, with vomiting once or twice; then a pain is felt in the right iliac fossa, which is generally tender and rigid, and a tumour is eventually found here.

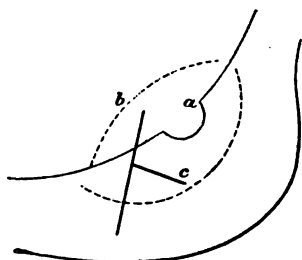
Could it be ruptured tubal pregnancy? This girl is of an age at which she might possibly be pregnant. She is an unmarried woman of 20, but the history was quite different from that of a ruptured tubal pregnancy. In the first place, the latter condition rarely occurs in a woman who has not had a child previously. Generally there has been some lesion in the tube before, due to a previous confinement; then the pain is pelvic, not epigastric. Again, a patient with tubal pregnancy is the subject of hæmorrhage, which makes her faint, and such patient does not get the quickly supervening acute peritonitis which this girl had.

Another perforation which may be mistaken for that of gastric ulcer is perforation of the duodenum, close to the stomach. Fortunately it is not of very great importance that you should diagnose between perforation of the stomach and of the duodenum, because the treatment is the same in each case. I will say this, however, that perforation of the duodenum is commoner in middle-aged men, and of gastric ulcer in young women, or in men between 50 and 60.

There was, then, quite sufficient to enable a diagnosis to be made here with ease, and, having done this, what naturally followed? Assuming that the patient was in a fit condition to bear a prolonged operation, we had to open the abdomen, find the perforation, close it by suturing, if possible, and thoroughly clean the belly.

This girl improved so much by stimulation with brandy and strychnine, that what at first seemed quite unfeasible became feasible, and when she had been in the hospital a little time I decided to operate. Chloroform was given, which is much preferable to ether in cases of this kind, where the distended belly encroaches on the diaphragm, and pushes it up into the thorax, and thus interferes with breathing. A median incision, rather more than 4 inches in length, was made, commencing an inch below the ensiform cartilage. It was noticed that the deeper tissues, especially the sub-peritoneal tissue, were very œdematous. On opening the peritoneum, there was a great gush of gas and fluid (altogether some pints of fluid escaped), and towards the end this fluid became purulent.

The tension in the belly was so great that when the peritoneum yielded to the scalpel there was a fizz of gas and fluid, the latter shooting up 18 inches into the air. As soon as the peritoneum was freely opened, the liver lifted up, and the dirty, purulent material sponged away, the perforation was recognized, located close to the lesser curvature, and about midway between the pyloric and cardiac ends of the stomach, and the size was about that of a shilling. Having found the perforation, the next step was to get out of the stomach all the food, bile, and gastric secretion which remained in it, because these contents were gradually pouring out of the perforation. Therefore, by means of a tube, and by palpating the stomach, I expressed through the perforation all the fluid and semi-digested food, and cleaned it away. Having done that, I found I had not sufficient room to do what I wanted—viz., to stitch up the perforation, so I added to my vertical incision a transverse one towards the left, about $2\frac{1}{2}$ inches long, which gave me easier access to the hole. Thus:—



a, perforation; *b*, longitudinal incision; *c*, lateral incision, to permit of stitching up. The dotted lines indicate the sutures.

The liver was held up firmly by retractors, and a first row of seven interrupted Lembert sutures of silk was inserted. Some of these cut out of the stomach wall, which was rather soft, and I had to put in others. When these seven sutures were tightened, and the edge of the perforation folded in, it was almost closed, but not quite; there was still a little leakage. Therefore I had to put a second row of five sutures outside the others. The next duty was to clean the abdomen, and that, perhaps, was the most difficult thing of all—to thoroughly wash out the whole belly cavity. To secure that this irrigation be effective, I think it is necessary to make an opening above the pubes. It is almost

impossible, by pouring water in at the top of the abdomen, to thoroughly wash out all the parts, especially the lumbar and the iliac fossæ and the pelvis; therefore I made another opening above the pubes, and washed out the whole belly cavity with a saline solution—0.6 per cent. of ordinary salt in warm water. By putting the irrigating nozzle in at the upper incision, and working down, and then by thrusting the nozzle in below and getting my hands in partly above and partly below, I was able, as I believed, to thoroughly clean the belly cavity. But it is evident from the post-mortem notes that I missed one place, between the liver and the diaphragm. Having irrigated the belly through these two openings, I put a drainage-tube in the lower one, into the pelvis, and the upper one I partly sutured and partly packed with gauze, right down to the place that I had closed in the stomach. The gauze there acted as a drain. During the course of the operation, which occupied 1 hour 25 minutes, the pulse, which was bad at first, improved as soon as the tension in the belly was lessened. That fact is rather important. Even at the end of the operation the pulse was better than at the beginning. There was no doubt that the girl was suffering greatly from the extreme tension in the belly—due to the presence of fluid and gas. She was, of course, put back in the ward as quickly as possible, but she was more collapsed half an hour after she left the table than she was during the operation. Brandy was given by enema, and morphia was injected about two or three hours afterwards, and 4 minims of liquor strychniæ were given hypodermically every four hours. By this, and the repetition of nutrient enemata every four hours, the patient was just kept going. I cannot say that her pulse improved in quality, but it did not get worse, and remained at about 140 to 160 throughout the next forty-eight to fifty hours, when she died.

When the patient had struggled through the first twenty-four hours, I began to think that there was perhaps a shade of hope that she would get well, and the next morning I still had that hope, because the bowels had acted spontaneously; she had several times passed flatus, and the abdomen, which was convex when we put her back after the operation, though still rather full, had now become concave, indicating that the intestine had recovered the power of emptying itself, and that the

peritonitis was subsiding. Its subsidence in the region occupied by the intestine led to the hope that it was subsiding elsewhere. Nothing is more characteristic of improvement in peritonitis than that the bowel is able to expel its contents, and that an abdomen previously convex becomes concave, and at the same time becomes movable in the act of respiration, instead of remaining, as hitherto, fixed and rigid. However, like a great many other things of this kind, the end was to be disappointing, the girl dying about fifty hours after the operation.

Now as to the post-mortem, which I will sum up briefly.

First of all, on opening the belly, we found very little evidence of peritonitis anywhere in the region between the margin of the stomach and the pubes; things had improved very much indeed. On removing the intestine there was some lymph found in the pelvis, but not any considerable amount. Down by the hepatic flexure of the colon, there was adherent omentum and some lymph, but on lifting up the right costal arch we found a considerable collection of pus, between the upper surface of the liver and the diaphragm. It was thus quite evident that we had not thoroughly cleansed this district by the process of irrigation. One should always endeavour to learn something by one's mistakes, and I cannot help feeling that this was an unfortunate thing, and that it had, at all events, something to do with the bad result which attended the operation. I am not prepared to say she would have got well if that space had been thoroughly cleansed, but undoubtedly the failure to do so was an unfortunate incident. The truth is, these patients are so ill during the operation that one is most anxious to get them finished with and put back into bed. Although that is a very proper and legitimate feeling, one must be careful not to give it too much play. It is most important that everything that is done should be done thoroughly; above all things, irrigation should be complete.

Now what about the stomach? We took it out, and found a very large ulcer in it, about $2\frac{1}{2}$ inches long by $\frac{3}{4}$ inch broad, lying partly on the anterior and partly on the posterior walls, like a saddle, across the lesser curvature. On the posterior surface it was adherent to the pancreas, and it had only perforated through at the anterior portion.

Some of the sutures had given way, and an escape of fluid was taking place through the stomach. It seems very likely, however, that the breaking down of the sutures was due to the digestion of the edge of the perforation rather than to the simple cutting out of the stitches.

What is the lesson to be learnt from this case? I have already considered the diagnosis; now to consider the treatment. Here I wish to lay great stress upon the immediate treatment which should follow the symptoms of perforation. There is pain and collapse, and the tendency, of course, is to give opium to relieve the pain, and brandy to combat the collapse. Both these things are very properly given, but the opium should be given rather in the form of morphia injected under the skin, and the brandy should be given per rectum, because we need to particularly avoid putting anything into the stomach. Why? Because it would probably find its way out again, or some of it, through the perforation into the belly cavity, and if it *fortunately* happens that at the time of the perforation the stomach is nearly empty, the most UNFORTUNATE thing we can do is to put anything into it which can increase the amount of extravasation. In addition to the administration of stimulants and opium, put the patient in bed and keep her in a *horizontal* position; don't allow her to sit up, and so permit the fluid which may be so far well localized to run down all over the intestines. What I have just told you as to treatment applies to the first hour or so after the perforation, after which period the collapse will have passed off, or will have begun to subside. The next point is to decide whether you will advise an operation to be done to close the perforation and clean the abdomen, and prevent the patient dying, as otherwise she would be very likely to do, from peritonitis. Whatever decision is arrived at, it is still equally important that the stomach be kept empty. If the friends consent to an operation, arrangements for carrying it out should *at once* be made, so that it can be commenced as soon as the patient has recovered from her collapse, but not before, otherwise she may die on the table. It is well to remember that the operator in such a case has a long operation before him, even under the best possible circumstances. He must be fully prepared *beforehand* with everything he *may* want. The favourable time for operating in these cases is three to five or six hours

after perforation has occurred. If you can begin in the third hour, so much the better, but you can scarcely get your preparations completed earlier.

The next point is to make a free opening in the abdomen; don't make a little hole which will scarcely admit two fingers; four inches at least; five or six if you like, and at the middle line is the most convenient place. Then try and find where the perforation is. But remember that if it is an early case there may be very little extravasation, very little evidence that there has been perforation. Lift up the liver and look under that. Between the liver and the stomach you may see a little food material which has leaked out, which will enable you to find the perforation. Don't let any of the food which is lying between the liver and stomach escape lower down and soil the colon; clear it out with sponges at once. If, on the contrary, you find there is widespread extravasation, you will not be able to mop it up with sponge, but must resort to thorough and complete irrigation. Irrigation should be reserved for cases in which the extravasation is on a large scale, because irrigation in slight cases would result in the particles of food being carried to new points.

Then, if possible, close the perforation by sutures, on the lines I have mentioned. If you cannot close it, the best plan is to pass a glass or rubber drainage tube from the incision down to the ulcer and just into it, and carefully pack all round it with iodoform gauze. This prevents any leakage taking place, except through the drainage tube; adhesions form around the gauze, and prevent any further extravasation. Under such circumstances the patient may recover, as has happened. But the best course is to close the hole if you can by sutures.

You will remember I made a transverse incision as well as a vertical one, and this should always be done if necessary, either to the left or to the right, whichever will give easiest access to the opening. After the operation it is, of course, necessary to feed by the rectum for several days, as anything put into the stomach would hinder the closing of the aperture in its walls, or would leak out, or might set up vomiting, and result in some of the material being squeezed out of the perforation despite your sutures.

Suppose the patient, or the patient's friends, will not have the operation carried out; or the condi-

tions are such that you cannot advise it; or again, suppose you are single-handed and have no reliable assistant, and so wisely do not attempt anything, as you need one, or better two, competent assistants besides the anaesthetist—what is then to be done?

You must do two things—continue to give the patient opium in some form, and keep the stomach empty, feeding only by the rectum. Under such circumstances, a few cases of gastric ulcer have recovered. If there is one point I would like to insist on in this lecture more than any other, it is the necessity of keeping the stomach empty, whether you are going to operate or not. A second point I would emphasize is this: if operation is to be done, it should be performed, to give it a reasonable chance of success, within the first six hours after perforation. I do not mean to convey that patients do not recover if the operation is delayed longer than that; they do recover when many hours have elapsed, but every hour's delay after the primary collapse has passed adds to the risk and danger. This unfortunate girl was operated on fifty-two hours after the perforation, so that the prospect of doing her permanent good was very remote.

CONGENITAL DISLOCATION OF HIP.

By C. GORDON BRODIE, F.R.C.S.,

Assistant Surgeon to the North-West London Hospital.

ROBERT B., a well-built and healthy boy of nine years, was brought to the North-West London Hospital by his mother because of a limp in walking. This had been noticed ever since his first commencing to walk, at the age of about 18 months.

When the boy stands naturally with the weight of the body borne on the sound leg (right), the heel of the left is noticed to be about $1\frac{1}{4}$ inch to 2 inches above the ground, placed closely against the malleolus of the opposite leg, the foot resting on the ball of the toes, which were everted.

On examining the left hip, the head of the femur could be distinctly felt, on movement, to occupy a position on the dorsum ilii directly above the acetabular cavity, its upper border being a little above the level of the anterior superior spine, the trochanter being correspondingly displaced upwards and outwards, but is behind the head. The head of the bone moves easily on the dorsum, and slides upwards and forwards nearer the superior spine when the weight of the body is thrown on to the left leg; the amount of this movement is about $\frac{1}{4}$ inch. Both the head and more especially the great trochanter are thrown into strong relief by bearing on the left leg, and the buttock much widened. The neck of the femur appears to be shortened and set nearly at a right angle with the shaft.

The left buttock is flattened, and the gluteal fold much higher than on the opposite side; the muscles appear somewhat atrophied, while the circumference of the thigh is about $1\frac{1}{2}$ inch less than that of the right thigh.

Progression is marked by a typical roll and swing, and when the left foot is placed flat on the ground there is marked lordosis with protrusion of the abdomen, the shoulders being thrown backwards, and a little scoliosis is observed.

The following measurements were taken :—

From anterior superior spine to	R = 26 in.
internal malleolus	L = $24\frac{1}{2}$ in.
Height of trochanter above Nelaton's	R = $\frac{1}{2}$ in.
line	L = $2\frac{3}{8}$ in.

The blocks illustrate most of the points extremely well, and were taken from photographs, the dotted lines showing the position of head and great trochanter when the weight of the body is not supported by the deformed limb (fig. 1), the lines the position assumed when the weight is supported by it (fig. 2).

The boy can easily walk a distance of five miles and does not get overtired; there was no history of any injury or disease of the parts; the labour, as far as I can ascertain, was natural, and without instruments.

Congenital dislocations of the hip should more properly be named congenital malpositions. They occur chiefly in females, the proportion to males being $7\frac{1}{2}$ to 1 (Lovett), and may be of all kinds,

though those on to the dorsum largely predominate. Congenital displacements other than those of the hip are very rare, and usually occur in subjects which have other deformities as well; while those of the hip occur for the most part in otherwise healthy individuals, though they may exist in conjunc-

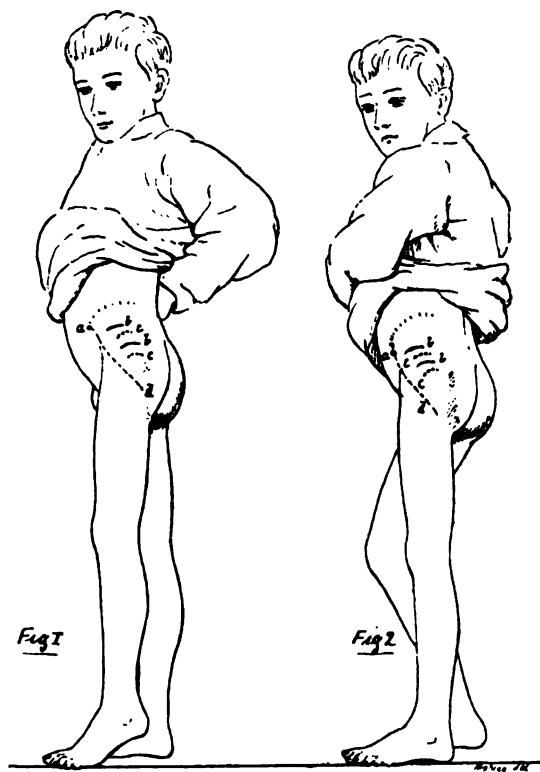


DIAGRAM I.

- a. Anterior superior spine.
- b. Position of head and great trochanter when weight of body is borne by the limb.
- c. Position of head and great trochanter when weight of body is borne by the right sound limb.
- d. Nelaton's line.

tion with other malformations. Putting together both Chaussia's and Parisee's records of autopsies upon new-born children, the result works out at one case of malposition in about 6000 cases examined, so that the condition can hardly be said to be a common one.

Several writers have dwelt on the part which heredity plays in the production of this malposition, and Dupuytren quotes the case of a girl who had eight relatives similarly affected, yet in many

instances there is no trace of other members of the family being afflicted with the same malposition.

Setting aside traumatism at birth, in a few recorded cases in which during the delivery of a breech presentation with a blunt hook the head of the femur was felt to slip out of the socket, and which are impossible afterwards to distinguish from true malpositions, the theory which best accounts for the position of the head of the bone is that of deficient development of the acetabulum.

Paletta first thought that it was due to excessive ossification of the Y-shaped cartilages filling up the cavity, but Growitz, in a series of preparations, came to the conclusion that it was due to deficient ossification of the same, and points out that in luxations of the joint, where the head of the bone is displaced and there is no physiological use for the joint, the same conditions prevail.

Mr. C. B. Lockwood exhibited at the Pathological Society (vol. xxxviii.) two foetal specimens which showed clearly that the rim of the acetabulum might be deficient; in one the head was lying upon a flattened surface in situ, and in the other the head was displaced on to the dorsum ilii. Both foetuses were males, breech presentations, and assumed a position after birth with the thighs flexed and the legs extended on the thighs (the quadriceps extensor muscles being too short to allow any other position). Both had the rim of the acetabular cavity absent, a capacious capsule attached where the rim should have been, and a long ligamentum teres. In the foetus where the head of the femur was not displaced it was normal in shape and size and lay upon a flattened surface, while in the other both heads were small and irregular, and displaced on to the dorsum ilii beneath the anterior superior spine of the ilium; the capsule being pouched upwards and separating the head of the bone from the dorsum ilii.

From these and other sources I gather that the acetabulum of new-born children afflicted with this malposition is shallow, without a rim, narrow and elongated, with the femoral head a little larger than the cavity (Young), with a capacious capsule and elongated ligamentum teres.

In the adult pelvis the acetabulum will be found to be smaller than usual, triangular in shape, with the base at the cotyloid notch and apex at the ilium very shallow and, but for a slight ridge marking the attachment of the capsule, rimless. The

accompanying sketch is taken from specimen No. 1774B in the College of Surgeons' Museum, and illustrates the foregoing remarks. The acetabulum in this case is almost filled up by a bony growth



DIAGRAM 2.

Left side view of pelvis. Showing the femur not much displaced, but resting on a shallow depression without a rim.

at the back portion, but is deeper in front, where the cotyloid notch is.* (I am indebted to Mr. Targett for his kind permission to have this specimen drawn, and also for permission to have it out of the museum to-night to show the Society.)

Now it is well known that continual movements are necessary for the formation and development of joints, and should the head of the femur be misplaced upon the dorsum ilii the loss of its upward pressure against the iliac segment of the

* The right side of this pelvis shows the femur displaced considerably upwards, while on the left side the femur is still nearly in situ, though resting on a shallow depression, and thus corresponding in some measure with the first of Mr. Lockwood's cases.

acetabulum would permit the bone to go on developing and fill up the cavity of the acetabulum just as it has done in the specimen before us; this may be a factor, and no doubt is, in helping the triangular adult shape of the cavity, but in view of Mr. Lockwood's specimens there can be no doubt, I think, that the condition which allowed the former to escape from its normal position was due to a deficient development of the cavity in the first place (especially as he has pointed out that the acetabulum is formed by a heaping up of cartilage

head the pelvis is slung up on either side of the thickened capsular ligaments and the muscles attached to the great trochanter somewhat after the fashion of the old C-springed coach. Even the ilio-psoas muscle appears to bear some of the weight, for the groove in which it lies as it passes out of the pelvis is usually much deepened.

While hung in this way the upward pressure of the femora upon the dorsum ilii cause them to be approximated; the tuber ischii, on the other hand, are much wider apart. The femoral heads are

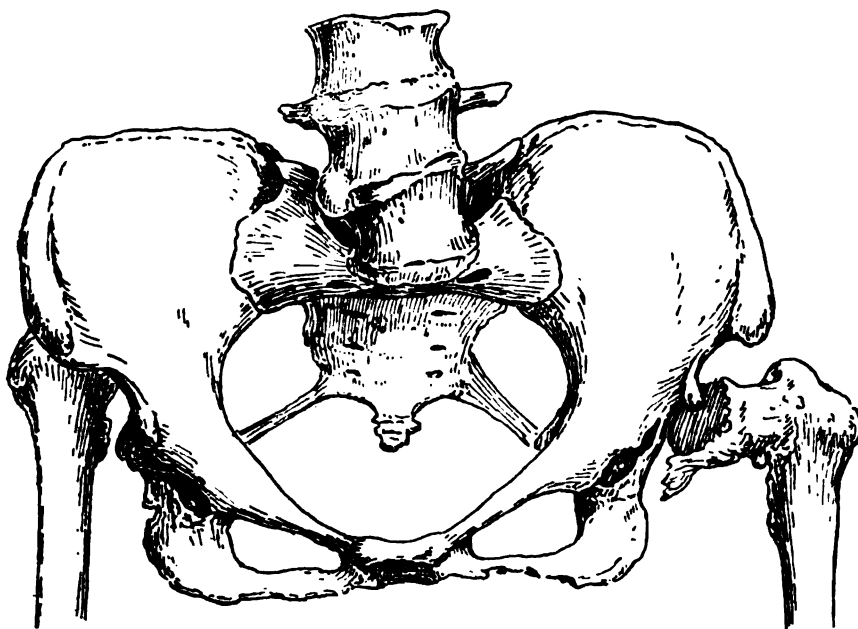


DIAGRAM 3.

Front view of specimen No. 1774 B, College of Surgeons' Museum.
To show the general aspect of the pelvis.

round the head of the femur, and not by pressure), which may perhaps be combined with some abnormal position of the limbs in utero.

Where the head of the bone presses on the dorsum ilii there is usually a roughened shallow depression, and upon the position of this depends the amount of tilt which the pelvis has. Should it be directly above the cavity, the angle of the pelvis remains much the same, and there is but little lordosis; should, however, the depression be more posterior, the pelvis tilts, and severe lordosis occurs.

On account of the new position of the femoral

smaller and irregular or flattened in shape, and the necks shortened and set less obliquely on the shaft.

The treatment of this condition was unsatisfactory until Lorenz introduced his operation, and appeared to be mechanical rather than operative.

Resection has been tried, but out of some sixteen recorded cases only three were good, five moderate, and the rest bad, and when the result was good the limbs were much dwarfed by the amount of bone cut away. De Paoli tried to excavate the acetabulum and then pegged the head of the bone, but it did not leave a good result.

Hoffa, starting on the assumption that all the muscles passing from the pelvis to the thigh were



DIAGRAM 4.

Right side view of pelvis, No. 1774 B, College of Surgeons' Museum.

- a. Acetabulum.
- b. Points to ridge marking position of acetabular rim.
- c. Depression, where head of femur rested.

shortened, and so caused the obstruction to replacement, devised the following operation. In children over six he first divides the hamstrings, adductors, and soft parts in front of the thigh, as far as possible, down to the capsule; then by means of a Langenbeck's resection incision he lays open the capsule, dividing subperiosteally the muscles attached to the great trochanter, and with a raspatory clears all the muscles from the femur as far as he can reach, gouges out the acetabulum, and by strong flexion and adduction, replaces the head of the bone. In children under five he exposes the capsule first, strips the femur, and then replaces the head of the bone by flexion and direct pressure, afterwards extending the limb, and so gradually stretching the long muscles. He does not recommend any operative treatment above the age of ten, and as he only claims to minimize the limp by a serious and troublesome operation, it is hardly an encouragement to perform it, especially as the ultimate results are said not to be so good as the immediate ones.

Lorenz, on the contrary, states that the pelvi-trochanteric muscles are lengthened rather than shortened, whereas the pelvi-crural are shortened, the degree depending on the amount of displacement of the femur; hence it is superfluous to divide the *former*, and as they are the muscles which brace the head of the femur against the hip bone, it is still more important not to divide them. He also shows that in children of 6 or 7 years with more than $1\frac{1}{2}$ inch of irreducible shortening, the muscles attached to the tuber ischii must

not be interfered with, lest the sciatic nerve be stretched and palsy or persistent neuralgia ensue. Lorenz divides his cases in three classes:—

1. Those in which the head of the bone is easily replaced, as in children from 3 to 5 years.
2. More severe cases between the ages of 6 to 8 years.
3. Severe cases between the ages of 9 to 12 years.*

For the first two classes, with the exception that forcible extension by a screw is used in the second class, he makes an incision from the anterior superior spine of the ilium along the outer border of the tensor vaginæ femoris for about three inches. The fascia is divided in the same line then running backwards along the anterior border of the gluteus medius. The tensor, sartorius, and rectus are drawn forward and the capsule exposed and divided, the acetabulum gouged out, and the head replaced. This does not do for the third class, where previous extension by a weight (up to thirty pounds) and pulley is necessary for a fortnight, and when the head can be reduced, open the capsule.

Special attention must be paid to the formation of the new acetabulum, which must resemble the normal one as closely as possible, taking care always to have the upper border left sharp; this part of the operation is usually accompanied by very troublesome hæmorrhage, and trouble should be taken to fit the head of the bone well into the socket.

If the head of the bone remains in the acetabulum when the limb is lying straight or slightly adducted the result augurs well. Do not stitch the capsule, and have a central drain in the wound; aseptic dressing is next applied, and a light plaster from the axilla to malleolus, fixing the limb in slight abduction.

Lorenz allows the children to get up on the fifth or sixth day, and at the fourth week removes all plaster and uses gymnastics and massage, but finds no use for any supporting apparatus afterwards.

However perfect the result, there is usually a slight limp left, which gets less as time goes on, and in cases operated on in the first two years of life is scarcely appreciable. There is also some slight shortening of the femur from the shortening and alteration in angle of neck, and even should ankylosis occur it is found to be incomparably better than the movable swing joint; out of 100 cases there were no deaths, and 99 healed without any reaction.

Mechanically, a good deal can be done by extension, and the following cases are reported in the American journals, which, if the results are lasting, are extremely good.

Bradford reports a case in which a girl of three years was kept for several months on her back

* "Annals of Surgery," vol. xxi.

with weights and pulleys until the head of the bone could be pulled down into the normal position; when treatment commenced the great trochanter was $1\frac{1}{2}$ inch above Nelaton's line, and at the end of extension it could be pulled down $\frac{1}{2}$ inch below it. He then put on a fixed apparatus which extended the limbs at a right angle to the trunk, thus relaxing the i'eo-femoral ligament and allowing the head of the bone to fall into its normal position. It also had the advantage of allowing the child to sit up and be carried about or taken out of doors (counter extension was made by means of a leathern abdominal and perineal band), and permitting a little passive movement in the way of rotation. At the end of two years the head of the femur was firmly fixed in the acetabulum, and could not be pushed out, even with sufficient force to push the whole body along the bed.

Buckminster Brown kept his patient, a girl of four years, for thirteen months under weights and pulleys, at an angle of nearly 90° with the trunk, and having brought the bone down to the natural position, he then forcibly rotated the knees outwards for a few hours daily, so as to produce pressure in the situation of the acetabulum; as soon as upward pressure did not move the bone, the patient was put in a wheeled chair, with the weight supported on a leather band and the toes just touching the ground to imitate walking, the weight being gradually allowed to rest on the feet, until after two years and three months from the original commencement of extension the child was able to walk, and judging from the photographs which are given of the case before and after treatment, the result appears perfect, the lordosis having almost entirely disappeared and the child standing upright.

Having in view the age of the child which I have brought before the Society, and his ability to walk well without fatigue, I hardly feel inclined to do much; if he were younger, I should certainly try extension for a time in order to relax the soft parts, and then perform the operation which Lorenz has described.

DEMONSTRATION OF CASES

AT THE

NORTH-WEST LONDON CLINICAL SOCIETY,

North-West London Hospital, November 20th, 1895.

DR. J. HERBERT STOWERS in the Chair.

Pleuritic Effusion and Pulmonary Cancer.

DR. GILL related the case of a woman aged 64, who, three-and-a-half years ago, had two very large

scirrhous growths in her right breast, one in the upper half and the other in the lower, but totally distinct, without any connecting link whatever, which he believed was very unusual. The late Mr. Marcus Beck, who had had great experience of cancer of the breast, had never seen such a specimen, and it was considered worthy of a place in University College museum. Since that time he, Mr. Gill, had removed a number of secondary growths. When operating, he removed the whole of the fasciæ, the whole of the axillary glands, and scraped the muscles carefully. So much tissue was removed that there was a difficulty in bringing the skin together, so that one corner had to be simply laced together. The skin became blanched and insensitive, but there was no suppuration; in fact, it looked dead, like a spongy graft. However, it became revived, new vessels grew in at the edge, and finally it recovered, without leaving a cicatrix. He, Dr. Gill, had on four occasions during the last three-and-a-half years removed secondary growths just over the line of the axillary glands, but only on one occasion was the growth down on the intercostal muscles. Recently the patient under consideration was seized with very bad breathing and could hardly get about. He then found that the left side of the chest was absolutely full of fluid, right up to the second rib. As it was inconvenient then to operate, he postponed it. About a month ago he was sent for because the patient was dying. In moving about she had been seized with intense dyspnoea, and on his arrival she was black in the face, and cold; no pulse was to be felt, and she seemed to be sinking fast. Fortunately, he had his aspirating instruments with him, and he removed over a pint of fluid. The patient rallied well, but, as he anticipated, the fluid re-accumulated, and three days later he aspirated again, and as no bad results accrued before, he this time removed a quart. Once again the fluid re-entered, and he was called in again, when the patient was moribund; again he drew away a quart of fluid. Since that there had been no further effusion, and that was a month ago. That day he examined her and felt scarcely any fluid at the base of the lung; the dulness had disappeared, and now the patient was up and about, and seemed to have a further lease of life. A curious fact was that a growth had now occurred on the opposite side to the former one; but though the growth removed was an extensive, old standing one, there was no sign of trouble on that side. She was free except in this left lung; he said lung because there was no tenderness along the spine; if the glands in the mediastinum were involved one would expect an impediment of the circulation. The lung was evidently saturated with new growths. It might have been interesting to members to examine the fluid for epithelial scales, but the fluid was too putrid to bring, and he had not drawn off any fresh.

In reply to Dr. Campbell, Mr. Mayo Collier, and Dr. Long, Mr. GILL said the growths were evidently simultaneous, and his opinion of their nature was confirmed by Mr. Marcus Beck, Mr. Pollard, and Mr. R. Johnson. Chiefly as an experiment, he put the patient on the same treatment as in ordinary pleuritic effusion—nightly doses of blue pill, diuretics, digitalis, and a potash mixture. There was no pleuritic pain.

Nævus.

Mr. BRODIE showed an infant with nævus, to illustrate the effects of two lines of treatment. The nævus was a very large one, partly over the fontanelle and partly over the frontal bone. He cut out the nævus six months ago, and for a few weeks matters progressed very well; but six weeks after, it commenced to grow again rapidly, though he thought he had got well below it. When it had reached its former size, he tried electrolysis, by means of gutta percha coated needles. This had stopped the growth, and though it might need one or two more touches, he thought it illustrated well the superior result from electrolysis.

Dr. CAGNEY asked why needles coated with gutta percha were used. This coating seemed to him to introduce the only source of danger in the process, because of the difficulty of rendering that material aseptic. He presumed the idea was to protect the skin, but his experience did not show him either that there was any need of that, or that insulation of the needle did in fact diminish the scar. Quite the contrary. If such needles had any good effect in Mr. Brodie's experience, the members would be glad to hear of it.

Mr. JACKSON CLARKE said he sometimes used an insulated needle coated with shellac varnish to preserve the epidermis, as occasionally there was a tendency for the vessels to thrombose. But he had not noticed any difference in results. He agreed with Dr. Cagney that insulation was not necessary, but if such were desired, shellac varnish was free from the objection which might be urged against gutta percha.

Dr. STOWERS expressed his pleasure at the success of the electrolytic treatment, which he recommended because he had seen such good results therefrom. He thought operation with the scalpel should be avoided wherever possible for the three reasons that:—in electrolysis—(a) the danger was considerably lessened; (b) the scarring was materially reduced; (c) that it was not necessary to administer an anæsthetic; which in children especially was an important matter.

Mr. BRODIE said he had used platinum needles; steel and other metals he found to leave considerable marks on the skin. The layer of vulcanite he used was very thin. He plunged the needle boldly in, and had no trouble with it afterwards. With the bare metal he found the skin sloughed just round the needles.

Congenital Dislocation of the Hip.

Mr. BRODIE showed a boy, 9 years of age, the subject of congenital dislocation of the hip, who had been born naturally, after a perfectly normal labour, and without instruments. (Mr. Brodie has favoured us with a full account of this case, with diagrams, and it will be found on p. 84 of this number.) Mr. Brodie said he would like to take the opinion of the Society on the question of treatment. The boy could walk $5\frac{1}{2}$ miles without feeling over-tired, and he was therefore much inclined to leave well alone. He had caused to be brought a museum specimen (which was presented by Mr. Adams), derived from a woman of 60 years of age.

Mr. JACKSON CLARKE thought the present a most interesting case in many ways; on account of the rarity of the affection and the extreme difficulty of successful treatment, as well as because of the great amount of attention the disease had attracted during the last three years. It was most important to try and gain an idea of the exact condition prevailing at birth, and with that view he had brought a dissection of a double congenital dislocation which he found in a fœtus that had spina bifida and slight hydrocephalus, and in which he had been able to fix the exact position occupied *in utero*. The uterus contained several fibroids; and he believed the affection was due to a cramped space in the uterus. To say that it was due to insufficient development of the acetabulum was not stating a cause, but only one of the phenomena. He controverted Mikulicz's contention that such cases were normal at birth; also his view of the pathology. He agreed with Mr. Brodie that operation on the boy before them was out of the question, and he thought his surgical friends would also say that was the best course.

Mr. BATTLE said the case differed somewhat from those usually seen, because the head of the bone was placed further back, more on the dorsum, and did not point in the usual direction. Regarding the rolling gait, he had known cases of double dislocation taken in hospital for pseudo-hypertrophic paralysis, in which disease, attitude and walk were exactly similar. The position of the bone in this case was exceptionally good, and could not be improved upon by operation.

Plumbism and Monoplegia.

Dr. GUTHRIE presented a man of 52, the subject of lead poisoning, gout, and monoplegia. He had been engaged as a coach-painter most of his life, but did not use lead in his work quite so much as house-painters. He had had frequent attacks of gout, was in the habit of drinking a little beer, and was not alcoholic. Five months ago, whilst painting a carriage, he suddenly lost all power in his left arm, and his left leg was slightly affected; his face was drawn slightly to one side; but he was able to walk home. He had no convulsions and no headache. He generally presented a some-

what flushed appearance, and certainly was not anæmic. There was nothing wrong with the eyes; the pupils were equal, and dilated and contracted normally; the discs were somewhat white, but there was no real atrophy of them. There was slight weakness in the left side of the face, but the tongue protruded fairly straight. The whole of the left arm was weak, particularly in grasping; there was fair movement of the fingers, but movement of the shoulder was considerably curtailed. Sensation was normal all over the arm, and there was no wasting; the supinator and triceps reflexes were exaggerated. He walked fairly well, but dragged the left foot slightly; the left knee-jerk was slightly in excess of the right, but there was no ankle clonus. His heart was not hypertrophied in any way; the impulse a little diffused. The radial arteries and those of the temple were tortuous, and rather hard. The urine was 10·10 sp. gr. and contained no albumen. He had the typical bright blue line on the gums.

As to the cause of the monoplegia, Dr. GUTHRIE gave his reasons for believing it to be due to the gout rather than to lead, though the question of the connection between gout and lead poisoning was, he said, a difficult one to decide. It was curious that in the northern counties, though lead poisoning is very common, it was very rarely that gout was associated with it. Some people said southerners drank too much bitter ale and the inhabitants of the north too much whisky. At any rate, the "four-ale" was liable to get tainted with lead by standing in the pipes all night. It had been said that both gout and lead poisoning led to the deposit of crystalline urates in the tissues and in the blood, and that both gout and lead had the same effects, but it was difficult to reconcile this with what he had pointed out. He thought the only explanation was that of Sir William Roberts, that people who had a tendency to gout were more likely to get lead poisoning than if they had no such predisposition. He did not think the lesion was likely to be in the cortex, because the patient had no cerebral symptoms at the onset, and because the symptoms were not so widespread as a lesion in that situation would produce. It was probably in the internal capsule, in the middle part of the posterior two-thirds, affecting the arm tract and the leg and face tracts, and he believed it to be a hæmorrhage there.

Dr. GILL spoke of the quantities of lead which could be taken into the system without producing acute symptoms, such as colic and paralysis. A man who was employed in grinding paints had been under his care and observation three years. Within an hour of starting work he could not see across the room for the fine powder in the air, and he declined to use respirators. He had suffered from most violent attacks of lead colic, but morphia had to be withheld because his urine was albuminous. He suffered from gout, and

undoubtedly had cirrhotic kidneys; he had also suffered from atheromatous vessels. His optic discs were unaffected, and no sign of paralysis was manifest whilst under his (Dr. Gill's) care. Part of the cause of his immunity might be that he was in the habit of taking nightly doses of sulphate of magnesia, and during his work, he (Dr. Gill) had persuaded him to drink sulphuric acid and lemonade, and to clean his teeth very thoroughly every time he went home. Regarding the connection between gout and lead poisoning: Twenty years ago, in Somersetshire, gout was very common in connection with lead poisoning in the cider-making industry. This cider was fermented in leaden vats. Since that time lead vats had been replaced by stone, and now lead poisoning was obsolete, and he believed gout was very much less in frequency.

Dr. CAMPBELL thought the patient had granular kidneys. He agreed with Dr. Guthrie as to the seat of the lesion, but not as to its nature. He believed it to be a case of thrombosis, not hæmorrhage, because in hæmorrhage there was loss of consciousness, but not in thrombosis. He agreed there was a relation between gout and lead poisoning; but as to gout itself, they should recognize that absolutely nothing was known about the pathology of gout, and the methods of treating it varied greatly. As to tortuous temples, it was a very interesting clinical fact, but he had seen such a condition in children of 7 or 8, and its absence had been noted in a man of 80.

Dr. GUTHRIE said if the lesion was thrombosis it would exist in a single vessel, and would produce cerebral symptoms. As the face was slightly, and the arm seriously affected, he thought it more likely that the cause was hæmorrhage in the internal capsule, but very small, and not sufficient to produce cerebral symptoms. He believed, with Dr. Campbell, that the patient had granular kidneys.

Epithelioma on the Back.

Dr. STOWERS brought a man of 76 years of age, with an epithelioma on the back, between the shoulders. There was no particular point in his history; the growth originated from a warty excrescence, similar to one just below the epithelioma, and the duration was said to have been six to seven months. The sooner it was removed the better; but he Dr. Stowers, would like to have the opinion of the members of the Society on it, particularly as it was in a somewhat unusual position. Before he saw it the case had been treated with poultices and iodoform powder.

Telangiectasis of the Face.

Dr. CAGNEY presented a patient, a woman of 40, to illustrate the successful treatment of telangiectasis faciei by the application of the galvano-

cautery. The patient had been under his care at the Hospital for Epilepsy and Paralysis. She suffered from tachycardia and enlarged thyroid, as well as the present disfigurement, and all the symptoms dated back a number of years. The result of treatment by the cautery was complete obliteration of the marks on the right side of the face. The left side was allowed for the sake of contrast to remain as it had been. There were scattered all over it large mulberry-coloured patches, some larger than a threepenny bit, and connected by radiating dilated capillaries. The disfigurement so caused was very great. The cautery left little or no scar if carefully used; and the difference between the two sides of the face as now exhibited was a speaking comment on its efficacy.

Intestinal Obstruction.

Mr. BATTLE showed a gall-stone which he removed from a lady in private. She was 64 years of age, very stout, and had had symptoms of intestinal obstruction of considerable severity for ten days. His first impression was that she had malignant disease of the upper part of the rectum. The abdomen was a good deal distended, and somewhat tender, but the patient's stoutness prevented anything being made out except that she was tympanitic. He found, on examining per rectum, that there was a hard, not very movable, mass, but he could not define it. The patient had brought up flocculent matter, and latterly had not been able to retain anything for any length of time. An incision was made in the left inguinal region, with the intention of opening the large intestine on that side. There was then found to be no distension of the large intestine, but of the small. He passed his hand down into the pelvis and felt the lump, and managed to draw it up into the wound, when he found it was made by a gall-stone. He could not displace it from its position in the gut, and therefore made an incision over it, the more readily as the mucous membrane had not lost its lustre, although it looked dark. He then found that the mucous membrane and muscular coats of the bowel were sloughing, and very offensive. Thereupon he secured the bowel outside and made an artificial anus. The patient was very exhausted, and died in six hours. The stone had become impacted in a loop of the small intestine, had dropped down into the Douglas' pouch, and was so low down that it could be felt from the rectum. The patient had no history of hepatic colic, and had not been jaundiced; but a friend said she had had previous similar attacks of pain.

Case of Bullet Wound in the Skull.

Mr. BATTLE showed the bullet which he had extracted from the head of the would-be suicide in a first-class carriage at Finsbury Park station

some time ago. He was admitted to the hospital in an unconscious condition, suffering from shock, with a hole in the centre of the forehead a little larger than the diameter of the bullet. The dura mater had been torn, and the anterior part of the frontal lobe on the right side had been damaged. He, Mr. Battle, was able to take away the bone and cleanse the wound and get out the bullet, without enlarging the original hole. There had been no rise of temperature since the operation, but there was some weakness around the scar, and a tendency to form hernia. He had excised the edge of the wound, so as to render it more thoroughly aseptic. The bullet was of a good size, and was fired at close quarters.

Mr. MAYO COLLIER, Dr. CAMPBELL, and Mr. JACKSON CLARKE offered some observations on gall-stones in general, and Mr. BATTLE replied.

Distortion of Palate and Face following Nasal Stenosis.

Mr. MAYO COLLIER showed two cases illustrating the ill-effects of chronic nasal obstruction on the growing skull.

The male patient with a history of nasal stenosis from the age of 12, and who was now 28, presented a face of peculiar disproportions. The upper maxillary bones were quite small, and so compressed to the midline, as to approximate the turbinated bones to the septum, and almost completely obliterate the nasal cavities.

The palate processes of the upper maxillary bones were almost vertical, a section of the palate being an acute angle.

The teeth were crowded, irregular, and laterally approximated, the molars on opposite sides not being more than an inch apart.

He now complained of deafness, constant hoarseness, and sore throat.

The second case, that of a girl *æt.* 18. The nasal cavities were completely obliterated, and the post-nasal space full of adenoids; the palate much the same as in the man's case, but the upper jaw was not so atrophic. She now complained of absolute deafness, with constant cough, hoarseness, dry tongue and throat, and disturbed sleep.

Dr. STOWERS exhibited a woman at 58 years, who had been the subject of Lupus for forty-two years. The cheeks and nose were involved, the latter being much swollen and red, and covered in parts with adherent crusts. The absence of the characteristic tubercles accounted for the long duration of the disease, and the very limited ulceration which had taken place.

Dr. STOWERS also exhibited a case of *Tinea Versicolor*, with microscopic specimens of the fungus. And, in addition, microscopic sections of—(1) Melanotic Sarcoma of the skin; (2) Rodent ulcer; and (3) the vesico-pustule of Variola.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 11, 1895.

A CASE OF GRAVES' DISEASE,

WITH UNILATERAL SYMPTOMS. TREATED BY
THYMUS FEEDING, ETC.

By P. WATSON-WILLIAMS, M. Lond.,

Senior Assistant Physician, Bristol Ro Infirmary.

THE case of exophthalmic goitre now recorded presents some features of special interest bearing on the recent views regarding the thyroidean origin of the symptoms which constitute Graves' disease.

Mrs. R., aged 35, came under my care at the Royal Infirmary complaining of dropsical swelling of the legs, for which she had been attended for nine weeks by the practitioner who referred her to me. She had first noticed transient swelling of the feet coming on towards evening, nearly a year ago.

On being interrogated as to other symptoms, she stated that she had noticed palpitation during the past three months, and for about the same time some fulness in the thyroid gland region. Temperature 99° F. The pulse rate was 110, the heart being normal in other respects. Pulse rate much increased on slight exertion. Respiration 28 to 38, lungs normal. There was considerable anasarca of the lower extremities, below the knee, and definite swelling of the upper eyelids, the latter not altered by pressure. Further, there was slight proptosis, well-marked Von Graefe symptom, hyperidrosis, fine tremors; flushing, and emotivity were not pronounced symptoms, though noticeable.

Catamenia regular, though deficient; urine 58 oz. daily, no albumen; no "bowel hurry" or diarrhoea.

Larynx normal.

As regards the thyroid gland, the right lobe was obviously enlarged, the left lobe was much less enlarged, but more than normally developed.

Further ocular symptoms were noted, viz. pulsation of the retinal arteries and veins of the right side only; shortly after admission the right pupil was larger than the left, both being dilated; facing moderate light they were 8 mm. and 7 mm. in diameter respectively.

VOL. VII. No. 7.

There was no paresis of the recti muscles, nor was the orbital frontalis paretic.

Weight 6st. 12lb.—Dynamometer. Right hand, 60 lbs., left hand, 30 lbs.

There was no family or personal history of neuromes, glycosuria, myxœdema, or rheumatism.

Before passing on to a brief *resumé* of the treatment pursued in this case, I would draw attention to the noteworthy fact that the Von Graefe symptom, the dilatation of the pupil, the enlargement of the thyroid gland, the hyperidrosis, and the dropsical swelling of the feet, were all much pronounced on the right side, while pulsation of the retinal vessels was observed on this side alone.

The excitement produced by one's visit to the ward always resulted in profuse perspiration of the right leg below the knee. On the anterior surface of each leg there was an extensive patch of persistent hyperæmia, much more pronounced and extensive on the right side, which corresponded to a diffuse brawny patch, reminding one of Hektoen's case, with a non-pitting œdematous patch in the same situation, which was found post-mortem to be due to an infiltration of mucinous substance.

On admission the patient was given Pilocarpin Hyd. gr. $\frac{1}{16}$, with three minims each of tinct. of cactus and liq. Fowleri in a mixture every four hours.

After five days, the patient having remained in bed, the œdema had greatly diminished, the thyroid swelling was distinctly less, the pulse rate varied between 96-102, respiration 32, temperature normal, the improvement being probably due to rest.

The tincture of cactus was then replaced by tinct. digitalis $\mathfrak{m}\mathfrak{v}\mathfrak{i}$. and v. of the liq. Fowleri given, and the galvanic current applied twice daily.

July 9th.—The condition of the patient remains much the same, the arsenic makes her feel sick. The mixture was discontinued. Pulse rather less frequent, feels better.

July 10th.—Ordered 3i. daily of sod. phosphate in mixture. After four days the only alteration in the patient's condition was a slightly diminished tendency to perspire. Pulse 92-99.

July 14th.—Ordered 1 thyroid tablet daily (= $\frac{1}{2}$ lobe each).

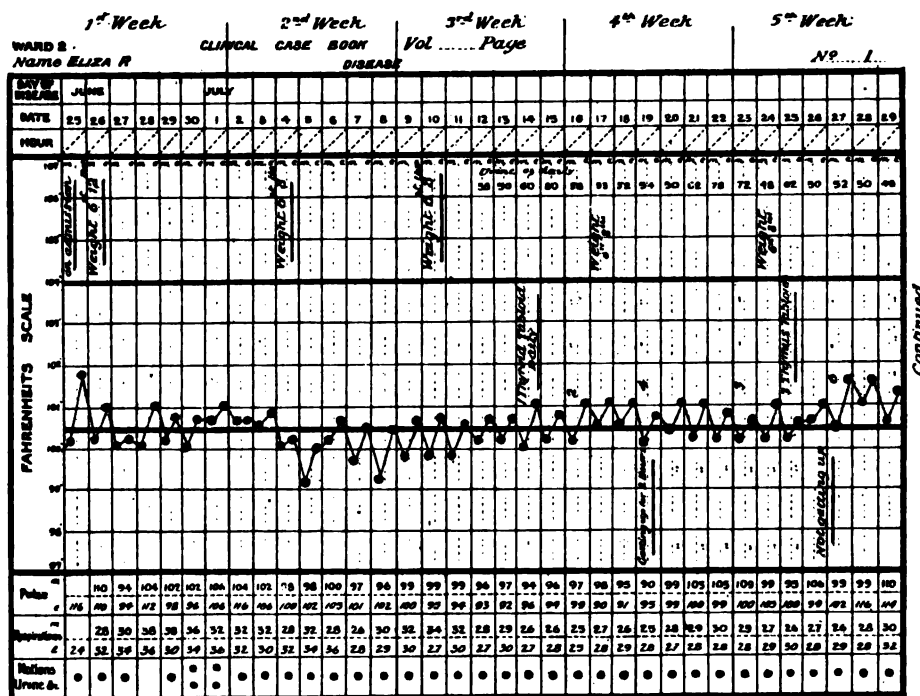
July 16th.—Ordered 2 thyroid tablets daily.

July 19th.—Ordered 4 thyroid tablets daily; these were continued till the 23rd, when only 3 were given daily, as the pulse rate had increased slightly, and the flushing and emotivity were certainly increasing again, and she did not feel so well. Urine was slightly increased in amount daily.

July 25th.—The thyroid tablets discontinued, and 3 thymus tablets given daily, increased to six

was given daily. The pulse fell to 98-120, but the rise temperature in the evening was the same.

Aug. 17th.—The thymus gland was stopped. Its administration had been attended with aggravation of all the symptoms. While on the thymus the patient complained much of the throbbing of the carotids, palpitation, etc., and declared she was more uncomfortable than she had been since admission. Proptosis became more pronounced. On leaving off the thymus tablets the symptoms subsided, but were again aggravated by the resumption of thymus in the form of fresh gland.



daily on the 27th, and continued to Aug. 2nd. By this time the pulse had risen to 118-122, and the temperature at night reached 99.4-99.6. The thymus was stopped, and in four days the pulse had fallen to 93-98 and the temperature become normal, and she felt much better again.

Aug. 7th.—Thinking that possibly the thymus tablets may have been impure, or had contained thyroid gland powder, some fresh raw calves' thymus (heart-berg) was ordered, one ounce daily. In five days the pulse had again risen to 132-136, and the evening temperature rose to 99.2.

Aug. 12th.—Only half an ounce of the thymus

Aug. 30th.—Patient has apparently recovered, to a certain extent, from the disturbing effect of the thymus gland administration, and is feeling much better than she was during the time she was taking it, the proptosis, Von Graefe sign, and palpitation being notably diminished, but the evening temperature still rises to 99.6.

Ordered mixture of Pilocarpin Hyd., cact. and liq. Fowleri again, as on admission.

Sept. 3rd.—Has improved greatly. Both pupils are rather large, and facing light the right is now 7 mm. and the left $6\frac{1}{2}$ mm. in diameter. Ordered Tinct. Bellad. $\mathfrak{m}\mathfrak{x}\mathfrak{i}$. daily, which was continued till

the 20th, when pills of Ext. Bellad. alcohol. grs. $\frac{1}{10}$ daily were given instead, and these increased gradually.

Oct. 7th.—She was taking as much as grs. $\frac{1}{10}$ of the alcoholic extract daily, and was feeling very much better and stronger; palpitation, emotivity much improved; no sweating even on excitement, pulse 90 98. The ocular phenomena not distinctly altered.

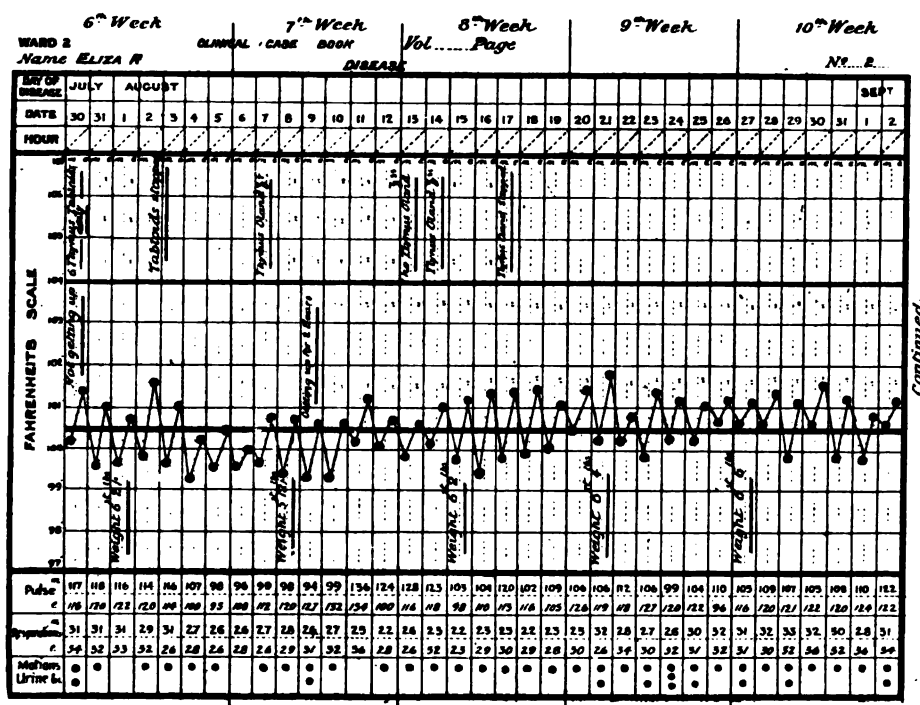
Oct. 11th.—2 c.c. of serum from a myxœdematous patient was injected into the buttock, the same repeated on the 14th. The only effect

thought she might be pregnant, and that that might account for the sickness.

Oct. 20th.—Made an out-patient—to take $\frac{1}{100}$ gr. of Atrop. Sulph. thrice daily.

Oct. 25th.—The dropsical swelling of the legs as bad as ever, but the palpitation and tachycardia, sweating and emotivity, tremor and goitre have not become worse again. Ocular symptoms in *stat. quo*.

A few days later the nasal mucosa of the septum and outer wall on either side was cauterized. The patient was not told the end desired, but



seemed to be to increase the pulse rate, and bring back palpitation and emotivity for a few days. (The myxœdema patients had been treated with thyroid extract, and greatly improved, so that myxœdema symptoms had disappeared, but no thyroid had been taken for several days prior to the serum being obtained.)

Oct. 15th.—Ordered pill containing—

Ext. Bellad. Alcohol. gr. $\frac{1}{8}$,

Pulv. digitalis gr. $\frac{1}{8}$,

Ferri Arseniat. gr. $\frac{1}{10}$,

five daily with food, but these were discontinued in three days on account of sickness. The patient

returning two days later, stated that her friends at home had remarked on the decided recession of the eyes. This was obvious, but the œdema and tachycardia were still present. Ordered—

Sparteïn gr. $\frac{1}{4}$,

Atrop. Sulph. gr. $\frac{1}{100}$,

Arsenite of Strych. gr. $\frac{1}{100}$,

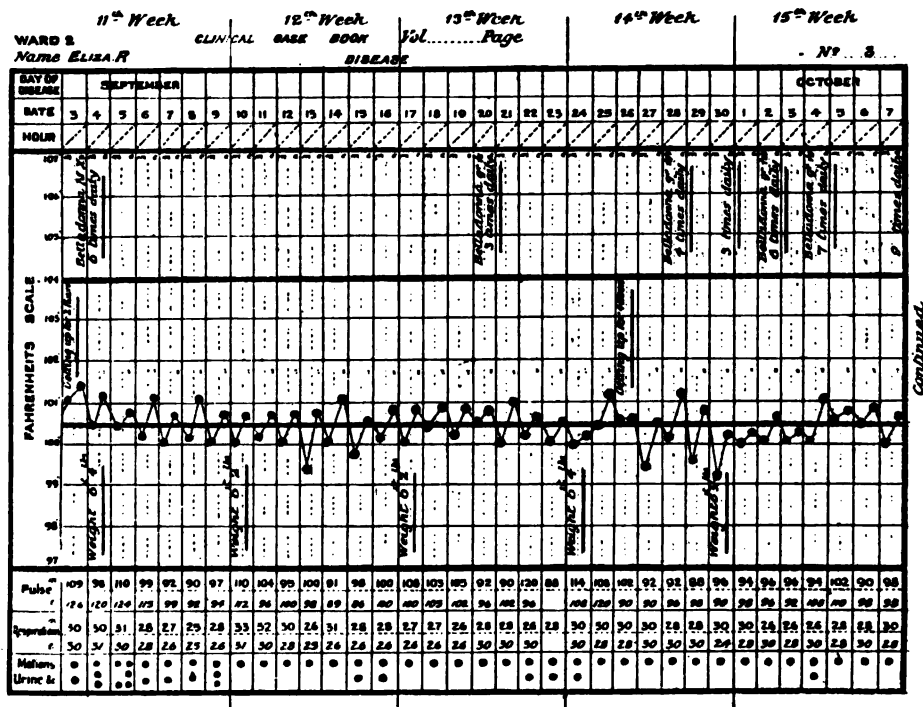
in pill, three times daily.

The tachycardia and palpitation and dropsy were much relieved in a few days, and the general improvement all round is distinct. It remains to be seen whether this will be maintained.

Remarks.—I have already drawn attention to

the chief point of interest in this case of Graves' disease, viz. the remarkable unilateral character of the main symptoms. How is it possible to believe that a general toxæmia by thyroid juice can produce such a condition? One could understand that one or more groups of phenomena might result from the action of a general poison affecting one side alone or more than the other side. We frequently observe this in diphtheritic and alcoholic neuritis, but when the unilateral character of the symptoms extends to ocular manifestations, enlargement of the thyroid gland, hyperidrosis, flushing

the spinal cord, in the heart muscle, in the muscular walls of the vessels, etc. But it is inconceivable that, between the lower organic centres in the medulla and the highest perceptive and volitional centres in the cortex, there are no intermediate or organic centres higher than those in the medulla, viz. subconscious-emotional or higher organic centres, which regulate and control the very complex organic phenomena of emotion. Such centres may perhaps be connected with the cerebellum, as suggested by Courmont. If such higher organic centres exist they are subject to disease, and



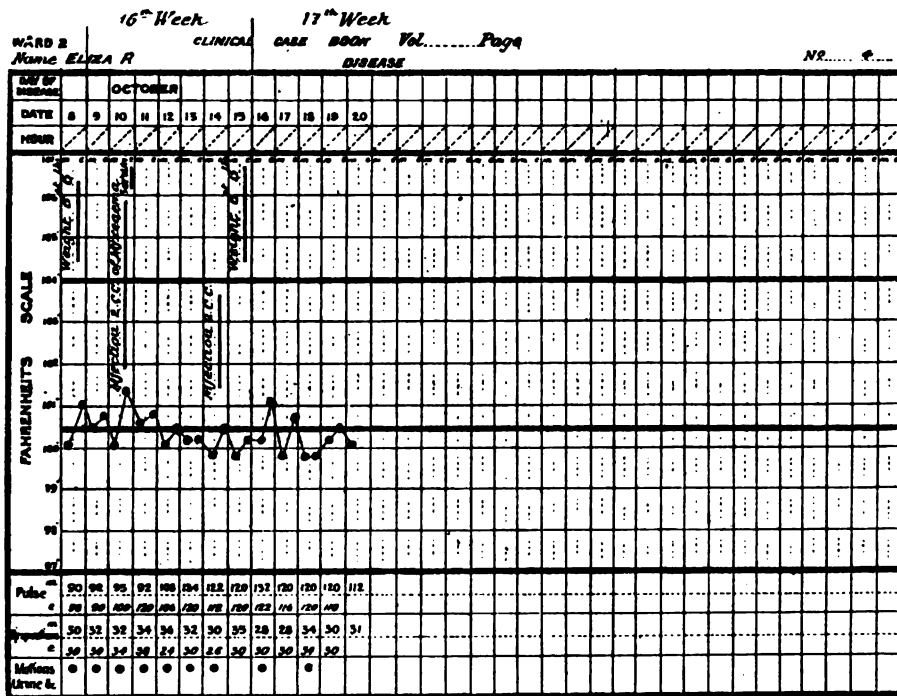
and œdema, it is difficult to avoid the conclusion that these symptoms are the result, not of a general toxæmia, but of a central nerve lesion. Fridenberg has been able to collect thirteen cases exhibiting unilateral ocular symptoms.

Any attempt to return to the exploded views that Graves' disease is a lesion of the sympathetic nerve ganglia, or due to medullo-pontal lesions is out of the question, although the medulla-oblongata does contain many centres presiding over complex co-ordinated organic phenomena, regulating and controlling in their mutual action and reaction still lower organic centres in

while on the one hand their disturbance will be attended by the highly complex organic phenomena corresponding to those normally associated with profound emotional disturbance, they will be accompanied by the still less understood conscious phenomena of emotion conveniently described as emotivity. Now sudden intense or prolonged shock or grief in the first instance inhibits the heart's action, slows the pulse, raises arterial tension, inhibits secretion of the skin, etc. But if sufficiently intense, or if acting on an inherently weak nerve structure, such a degree of molecular disturbance may be induced in the affected regions that death may be

caused either immediately or in a short period ; or if the disturbance is less marked, the affected nerve structures may be rendered more or less permanently parietic, with consequent secondary phenomena of emotional shock, viz. tachycardia, arterial dilatation with low tension, sweating, etc. Thus, from defective control, the accelerator nerve to the heart runs riot on the slightest provocation, while flushing, perspiration, and excitation are exaggerated by slight causes, such as the visit of a stranger, slamming of a door, and so forth, while, together with exophthalmos, ocular paresis,

myxœdema, but in small doses its primary action is observed, and just as it stops the excessive night sweats of phthisis, so probably it diminishes thyroid secretion. Yet its action in this respect is limited, since we cannot push the drug without inducing the secondary action which it is desired to avoid. Wherefore we have to resort to atropine, which can be pressed to its physiological limit. Digitalis or spartenie are well known to act beneficially in many cases ; they stimulate the parietic inhibitory cardiac nerve mechanism, and, like arsenic and strychnine, are most useful remedies.



and thyroid enlargement, they are more or less constantly present.

This group of symptoms is known as Graves' disease. The excessive thyroid secretion, when present, aggravates the symptoms which it did not cause, just as starch and sugar in the diabetic diet aggravates the disease which these common articles of food do not cause.

The action of atropine in Graves' disease is probably chiefly due to its lessening thyroid gland secretion, and is thus comparable to thyroidec-tomy. Pilocarpine in large doses is known to increase the thyroid secretion, hence its utility in

Another point of interest in this case is the result of administering thymus gland. Owen, Cunningham, and Kœher have seen beneficial results from giving thymus ; in fact, Owen and Cunningham claim that the exhibition produced a cure in their cases. In my case it only aggravated the disease, and behaved in every respect like thyroid feeding.

The experience of Enriquez and Ballet, and the investigations of Nobkine, as well as the recognized antithesis of myxœdema and Graves' disease, led me to try the effect of serum (obtained from blisters) of myxœdema patients in this disease, but

the conditions were not such as to enable me to draw any conclusion from the trial.

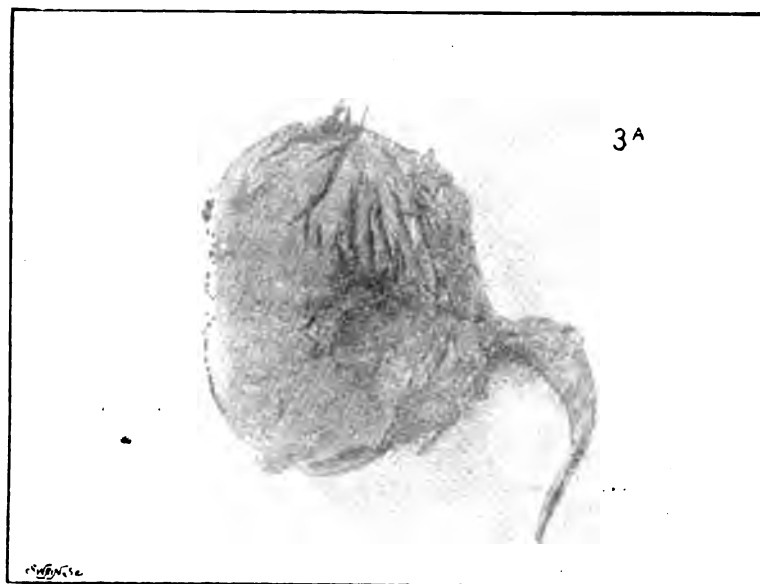
It is worthy of note that peripheral stimulation in the nose by galvano-cauterizing produced such a favourable effect on the proptosis. Similar results have been noted by Huck, B. Fränkel, Scanes, Spicer, and McBride.

Edema has several times been noted in Graves' disease, a rare symptom to which Maude has called attention, but it is, I think, unique to find that it occurred so early in the case, and for a long time was the predominant symptom.

ward in a child of five, now convalescent from nephrectomy, whose photograph I show you; another in No. 5 ward, under the care of my colleague, Dr. Barrs, recovering from an exploratory abdominal section, only however to die later from the disease, which is irremovable.

The notes of the cases which have been furnished by my house-surgeon, Mr. Robinson, are as follows:—

Case 1. G. B., aged 5, was admitted on June 17th for a swelling in the right lumbar region, presenting the characters of a kidney tumour. The swelling had been noticed for a year, and had been gradually and painlessly increasing in size.



Case 1, G. B., aged 5 (Fig. 1).

SARCOMA OF THE KIDNEY AND ITS TREATMENT.

Delivered before the students of the Yorkshire College,
July, 1895,

By A. W. MAYO ROBSON, F.R.C.S.,

Professor of Surgery in the Victoria University, and Senior
Surgeon to the General Infirmary at Leeds.

GENTLEMEN,—I have at present under my care two cases of sarcoma of the kidney, one in No. 10

There was no history of injury or tubercle and no alteration in the amount or character of the urine could be found.

Operation on June 20th, 1895, by an incision from the tip of the last rib to the crest of the ilium, opening the peritoneum, which was then stripped off the front of the kidney, the colon being carried inward with its peritoneal covering. The peritoneum was then closed by a continuous catgut suture, and the kidney brought to the surface, after ligaturing some attachments in the suprarenal region.

The ureter was found to be thickened ; and after ligaturing the renal vessels, it was stripped from the peritoneum nearly to the bladder before being divided, as a wormlike portion of the tumour was found to be passing into it from the pelvis of the

removed on the third day, and the stitches were taken out a week after the operation, when the wound was found healed.

The annexed photograph was taken after operation, before the child left the hospital.



Case 1, G. B., aged 5 (Fig. 2).

kidney. This is shown in the photograph of the tumour.

The cavity was well washed out, a drainage tube put through a small counter-opening in the loin, and the wound sutured in layers. The tube was

After note.—Seen on September 5th, and again on October 19th, in good health. No sign of recurrence.

Case 2. F. A., aged 18, a groom, was admitted under the care of Dr. Barrs on June 20th, 1895,

for swelling of the abdomen and general weakness. The illness began without apparent cause early in May, 1895, with pain across the stomach and vomiting, but there were no symptoms pointing to the kidney being the seat of the disease, and there was no alteration in the quantity or quality of the urine. He then for the first time noticed the swelling on the right side of the body, but after a week in bed he improved a little, though the swelling rapidly increased in size.

He was admitted on June 20th with an enormous semi-fluctuating and very tense tumour, occupying not only the left loin and left half of the abdomen, but reaching across the middle line. The extremely rapid increase of the tumour had caused such great tension that not only was the pain severe but the chest movements were impeded, and the breathing was considerably interfered with. The diagnosis rested between rapidly growing sarcoma and acute inflammatory trouble with deep suppuration. After consultation, an exploratory operation was decided on, in the hope of being able to relieve tension even if nothing more could be done.

On June 27th, an exploratory operation was performed through an incision of three inches, in a line vertically with the tip of the eleventh rib; the peritoneum was opened, and the compressed and empty colon found crossing the tumour. The peritoneum and colon were stripped off the front of the tumour and pushed inwards a little, the opening in the peritoneum being then closed by a continuous suture. The tense tumour was then incised, and a quantity of broken down growth and blood-clot evacuated, a drainage tube being left in.

As there was much infiltration of the neighbouring tissues, any further operative measures were impracticable, and the wound beyond the drainage tube was closed by silkworm gut sutures.

Thus far the patient has been very distinctly relieved by the operation, and he is much more comfortable, though unfortunately we know the relief can only be short-lived, as a microscopic examination of the portion removed shows the growth to be a mixed round and spindle-celled sarcoma.

After note.—The tumour continued to increase, and the patient died suddenly on July 23rd, a month after the exploratory operation, which had

not only given him relief, but had probably prolonged his life.

The disease in question affects specially two periods of life, the very young and adults after middle age, the clinical character and the structure of the tumour differing somewhat in each case; but although there is a proneness in infancy and in adult age, the case in No. 5 shows, if such proof were required, that the intermediate periods are not exempt.

Infantile sarcomata usually occur during the first seven years, and their growth is very rapid. The whole organ is generally involved, and, as in the present case, the suprarenal body may participate in the morbid process.

At first the organ retains its shape, and as there is little or no tenderness, the normal kidney outline can generally be made out by palpation. In this stage the growth is encapsuled, though in the later stages the surrounding tissues become infiltrated, as is well shown in the extremely acute case in No. 5 ward.

Hæmaturia seldom occurs, and in fact there is, as a rule, no alteration in the urine which would point to the serious nature of the disease in the kidney.

In these cases only one kidney has been attacked by the disease, but it is not uncommon, in the infantile form, for both to be affected.

Unlike some other enlargements of the kidney, this is painless; and in fact, beyond the steady growth of the tumour, and the gradual loss of flesh and strength of the patient, there are few symptoms to chronicle.

Thus you will see that the diagnosis has to be made from the physical signs, and, as a rule, the patient is only seen by the medical man when they are fairly well marked.

There will be a distinct tumour in one or the other side of the abdomen, which on bi-manual examination will be felt to extend into the loin, and to be capable of being pushed forward; it will at first be freely movable, and will descend when the patient is told to take a deep breath, though it may soon become too fixed to move on respiration.

There will be dulness on percussion in the loin as well as over the anterior surface of the swelling if the tumour be a large one, but as the colon passes in front of the tumour the degree of

resonance will vary, according to the presence or absence of gas in the large intestine. In large tumours, as in the one in No. 5, the colon is so compressed that the whole anterior surface is dull, whereas as shown in the case in No. 10, where the tumour was much smaller, the whole anterior surface was resonant. The contour of the kidney can usually be felt at first, and very frequently palpation will enable you to feel the rounded upper and lower ends of the swelling which will guide you in differentiating between a tumour of the liver and gall bladder or spleen above or any pelvic tumour below. Rectal or vaginal examination for the purpose of ascertaining the condition of the ureter will, as a rule in the earlier stages, give no information, but you will remember that in the case from No. 10, a worm-like process of the tumour had passed down the ureter almost to the bladder, so that here we might have obtained signs of ureteral enlargement.

These tumours may attain a great size, and though at first encapsuled, when they have gone beyond their capsule, they increase at an enormous rate and infiltrate extensively the neighbouring tissues. For instance, the case in No. 5 has attained an enormous size within a few weeks, partly due to growth and infiltration, but chiefly owing to hæmorrhages into the substance of the tumour.

I have operated on a sarcoma of the kidney in an adult, where the tumour was simply formed by one large sac with thick walls, the cavity being filled with broken-down clot and softened sarcoma.

These growths are composed of round and spindle-shaped cells in which tubules occur, and not infrequently striped muscle cells are interspersed, the tumour being then termed a myo-sarcoma or a rhabdo-myo-sarcoma. The progress of the disease is toward an inevitably fatal termination, though this is much more rapid in renal sarcoma in the infantile than in the adult variety.

In adults supra-renal tumours are not infrequently confused with kidney tumour. Some time ago I removed a large sarcoma which at the time I thought was renal, but afterwards the true kidney was found at its lower part almost enveloped in the sarcoma, which was of supra-renal origin. The patient, a lady of about 50, recovered from the operation, to die of a recurrence "in

loco" a few months later. Sometimes these supra-renal tumours attain an enormous size; one removed by Mr. Thornton weighed 20 lbs., and the patient recovered and was well a year after. True sarcoma of the kidney in adults is usually composed of spindle cells. The disease is unilateral and most common between 30 and 50. Hæmaturia is frequent, and the passage of clots gives rise to pain as does the loss of blood to anæmia. The duration is longer than in the infantile form of the disease, and death usually occurs from exhaustion or anæmia.

The diagnosis of enlarged kidney in the right side has especially to be made from tumour of the gall bladder. The following points are worth bearing in mind.

(a) Both an enlarged gall bladder and a normal or an enlarged kidney may produce a movable abdominal tumour.

(b) The history of an attack of jaundice is worth bearing in mind, though its absence does not disprove gall bladder enlargement.

(c) While an enlarged gall bladder can invariably be felt, a movable kidney unless enlarged cannot always be discovered.

(d) An enlarged gall bladder is easily felt on pressure in front, a kidney on bi-manual pressure.

(e) Both may vary in size, but diminution in a kidney tumour may be sudden and followed by a marked increase of urine voided.

(f) An enlarged gall bladder is as a rule very tense, hard, and pear-shaped. A kidney tumour is softer, and often presents the characteristic notch on its inner side.

(g) The movements of a gall bladder take place in the arc of a circle, the centre of which is a point beneath the right lobe of the liver, whereas a movable kidney has a wider range of motion.

(h) A movable kidney can be pushed down towards the pelvis and slips back beneath the palpating hand into its lumbar bed; a gall bladder moves with respiration, and not independently of the liver, in a vertical direction.

(i) Where the gall bladder is enlarged, the kidney can usually be grasped independently.

Having made your diagnosis, there are only two lines of treatment to consider: (1) General and palliative, which will consist in relieving symptoms as they arise, and (2) radical or operative, with a view to respite or cure.

In considering nephrectomy, unless the tumour be small I would not advise the lumbar operation on account of the want of room, nor would I advise the median incision or Langenbuch's in the linea-semilunares.

The operation, which you have seen me perform on several occasions, is through a ventral incision from the tip of the tenth rib towards the anterior superior spine of the ilium, or a little external to that; this opens the peritoneum just in front of the cul-de-sac, and enables you to ascertain the extent of the tumour and its attachments, after which the peritoneal opening is closed by a continuous suture; the peritoneal sac is then easily displaced inwards, carrying the colon with it, and enucleation is proceeded with as described in case 1.

In case of oozing from small vessels, a drainage tube is easily passed through a small lumbar opening, as in case 1.

The whole operation can be done very quickly, as the parts are well under observation.

In several of the text-books, notably in Mr. Bland Sutton's work on Tumours, nephrectomy in infantile sarcoma is spoken of as a useless procedure, first, because of the immediately fatal results of the operation, and secondly, because in those that recover from the operation, recurrence of the disease takes place within the year.

This is almost the truth, but not quite accurate. There have been cases operated on within this hospital where the disease has never recurred: in one case the child is living and well six years after.

Now, if we can save but a few cases, say, 5 per cent., it makes operation well worth pursuing, since nothing but nephrectomy gives the smallest hope of success; and seeing that the operation in the greater number of cases is followed by recovery and a more or less prolonged respite, I think it is our duty to place the exact facts before the patients' friends, and to be prepared and willing to operate if they elect to accept the risk in the hope of a slight chance of permanent cure.

A CLINICAL LECTURE ON HYSTERECTOMY.

Delivered at Guy's Hospital, November 4th, 1895.

By A. L. GALABIN, M.A., M.D., F.R.C.P.,

Obstetric Physician and Lecturer on Midwifery to the Hospital.

GENTLEMEN,—During the last five weeks we have had three cases of hysterectomy for fibroid tumour in my ward, therefore I thought a consideration

of a method which has been somewhat recently introduced for performing that operation, might form a profitable subject for my lecture. I propose to say very little to-day about the indications for hysterectomy, intending rather to discuss the technique of the operation itself.

You of course understand that it is only in a minority of cases of fibroid tumour that such an operation as hysterectomy is performed. The larger proportion of women having fibroids go on for many years without suffering serious symptoms, and attain the menopause, when the symptoms usually subside and the tumour tends to shrink. But the indication for operation varies according to the position in life of the patient. If a woman is in comfortable circumstances, and able to take care of herself as an invalid for a number of years, she will generally be more reluctant to consent to an operation, which involves a certain amount of risk to life, if the presence of the tumour itself does not threaten immediate danger. But if she has to earn her living, and the tumour disables her, she will be more inclined to run some risk for the sake of a cure. On the other hand, if a woman has severe menorrhagia, or pain, whatever her position in life, she will be anxious for relief, and the surgeon is justified in giving her the alternative of accepting the prospect of cure which the operation affords. Provided that she is a sensible woman, and is suffering from her tumour, I believe the right way is to explain to her, as clearly as can be, the amount of risk to life involved in the operation, and to let her decide for herself whether the amount of distress she suffers is sufficient to induce her to undergo the risk. It is obvious that, the more the mortality is reduced, the more the field of the operation can be extended.

There is another operation which should be considered before deciding on hysterectomy, viz., the performance of oophorectomy (the removal of the ovaries and Fallopian tubes). My own belief is that, in the early stages, a woman with a growing fibroid, and still a good way removed from the menopause, whatever her position in life may be, is wise in having oophorectomy performed, before the tumour has got so large as to be beyond the reach of that operation; that is to say, before the tumour has reached a much greater size than that of a uterus at four months' pregnancy. In the case of a comparatively small tumour in a patient who

has had no previous pelvic peritonitis, involving adhesion of the uterine appendages, I believe there ought to be only infinitesimal risk in this operation.

In my own experience I have not met with a case of death from oophorectomy performed under these circumstances. Generally, I believe the risk of death in such cases from the operation is put down at not more than one per cent. in the hands of a skilful operator. The case, of course, is different where there has been previous pelvic peritonitis, and especially if, as sometimes happens, the fibroid tumour has originated later than the peritonitis, because there is then a matting of the appendages, and it will be very difficult—probably impossible—to thoroughly remove the whole of the ovaries and Fallopian tubes. Under those circumstances the results of the operation on the growth, and on the symptoms produced by the fibroid, become uncertain; but if the appendages can be thoroughly and completely removed while the tumour is still small, in the great majority of cases menstruation is arrested from that time, although there may be some occasional bleeding. Afterwards the growth of the tumour is checked, and it tends to shrink, as after the natural menopause, although the process of shrinking may occupy a number of years.

But in many cases a woman who is suffering but little does not wish to undergo any operation until suffering commences. Again, many patients do not come for advice until the tumour has reached a larger size than that I have mentioned. When the tumour is much larger, then I think, as a rule, hysterectomy should be preferred to removal of the appendages, because in the case of a large tumour, the operation of oophorectomy becomes more difficult and dangerous, and in some cases impossible; at any rate, without turning the whole tumour out of the abdomen, for which a long incision is necessary. If it is successfully performed, it will take a long time for the large tumour to shrink, and the results on the tumour are more uncertain; also, the chance of menstruation continuing, or bleeding taking place, even after removal of the ovaries, is greater. Therefore, in cases of large fibroids, I think hysterectomy is indicated, if operation is required at all.

There are several varieties of this operation for

removal of fibroids, besides enucleation through the vagina, and these may be divided into three main groups:—

(1) First, there is what is called supra-vaginal hysterectomy, but which might perhaps be more rightly named supra-vaginal amputation of the fundus uteri. It is generally included in the term hysterectomy, although not all the uterus is removed; and is divided into two main classes: (a) with extra-peritoneal supra-pubic treatment of the pedicle, (b) with intra-peritoneal, or at any rate intra-pelvic, treatment of the pedicle. It is of one of the varieties of this second division that I mean to speak chiefly to day.

(2) Then there is what is termed total hysterectomy, in which the cervix is removed with the fundus uteri; and that again may be divided into three classes: (a) by the vaginal method, (b) by the abdominal method, in which the operation is almost entirely performed from the abdomen, and (c) by a combined method, in which the operation is performed partly from the abdomen and partly from the vagina.

(3) There is a third class of operation, in which not the whole uterus is removed, but the fibroid tumour. It is sometimes called hysterotomy, but more properly myomectomy.

This last operation is the one most suitable for certain particular cases, such, for instance, as that in which the uterus itself is but little enlarged, and without multiple fibroids in its walls, but has a large sub-peritoneal fibroid attached to it by a comparatively small neck. In that case the best treatment is to secure the neck by a ligature, excise the fibroid by a V-shaped incision, and stitch the peritoneum over the stump, so that no myomatous tissue is exposed.

Myomectomy is extended to other forms of fibroids, interstitial and even sub-mucous, but not always with the same degree of success. The plan in such case is to enucleate the fibroid out of the wall of the uterus, and close the opening with sutures uniting, as far as possible, the peritoneum over the gap. This has not yielded such favourable results as hysterectomy, but it finds favour with some, on the ground of preserving the uterus. I find that Martin's statistics of myomectomy, according to Obalinsky, give 18 per cent. mortality, as compared with certainly less than 10 per cent. in the various forms of hysterectomy.

I propose to first of all give the details of the mode of operating which was adopted in the three recent cases in the Dorcas Ward.

The operation is commonly known as Baer's operation, because it was originated by Dr. Baer, of Philadelphia. I believe, however, that priority has been claimed on behalf of a New York physician, Dr. Goffe, who published his first account of the operation in 1890. But it seems to me that Goffe's original operation was not the same as Baer's, because the only important novelty and improvement in Baer's operation is that no sutures or ligatures are used to tie uterine or myomatous tissue, whereas in the 1890 contribution, Goffe recommended that the ligatures securing the uterine arteries should be passed through the uterus itself.

The old method of performing supra-vaginal hysterectomy with the internal treatment of the pedicle was: in the first instance to secure the vessels by tying the ovarian and uterine arteries, or by putting ligatures round or through the cervix; then to excise the tumour by a V-shaped incision; after that to sew together the branches of the V so as to occlude the cervical canal, and then bring the peritoneum together over the surface. The results have been favourable with some operators, but in this country it has, on the whole, been thought that they have not been so satisfactory as from the external treatment of the pedicle. The stump formed of uterine tissue does not behave so well when embraced by ligatures or suture as does the stump of an ovarian tumour. There is a tendency for the uterine tissue to shrink and allow bleeding, or else become inflamed or suppurate in the grasp of the ligatures, and set up peritonitis.

The first point as regards hysterectomy is the preliminary treatment. The abdomen should be disinfected in the ordinary way as for any abdominal section, but in this operation, whether it is intended or not to open the vagina, it is desirable to disinfect, as far as possible, the vagina, and in some cases the cervical canal. This may be done by giving a douche of perchloride of mercury 1 in 2000 twice a day, or oftener, for some days previously to the operation, and for about 12 hours before the operation plugging the vagina with mercurial cyanide gauze. Some authorities recommend going even further than that, and using strong detergents to the part, and scrubbing

out the vagina with an instrument somewhat similar to a tooth-brush. Others attack the cervical canal by curetting that canal as a preliminary measure, or even burning out the lining of it with a Paquelin's cautery. All these proceedings, in some cases, as for instance in virgins with intact hymen, are naturally somewhat troublesome. For my own part, I think that in general they are unnecessary, and that, in the case of a virgin, even the preliminary plugging of the vagina may be omitted, it being sufficient to trust to the antiseptic douches.

In cases where there is any purulent discharge from the uterus, or in which the interior of the uterus has been treated by any electrical method or by caustics of any sort, the operator must do his utmost to thoroughly disinfect the cavity of the cervical canal and the uterus by washing with an antiseptic solution, even if curetting is not put in practice. For my own part, I am rather incredulous regarding the effect of curetting as a method of sterilizing the cervical canal. The membrane of the cervical canal is resistant as compared with that of the fundus uteri; it cannot easily be scraped away in shreds, and it has so many folds and convolutions that it is a very difficult matter to scrape the whole of it in such a way as to absolutely remove all such folds.

The next point as regards the operation is the position of the patient. Most modern authorities on hysterectomy recommend what is known as the Trendelenburg posture, in which not the feet of the patient, but her head is directed towards the light. Her body is supported on a sloping table in such a way that the knees are raised above the head, the whole being inclined at an angle of about 45°. In the operations recently performed, I have not adopted that particular position, but placed the patient in the ordinary way with the feet towards the daylight, having a lamp overhead which threw light into the pelvis behind the tumour as soon as it was turned out of the abdomen. But in many cases of hysterectomy the head could be turned to the light at a later stage with great advantage. In some of the early steps, such as during the separation of the bladder, the more convenient position is with the feet to the daylight, the table being turned round afterwards; and it is wise to have at hand the means for altering the elevation. For my own part, I

think it is generally enough to change the direction of the body without altering the inclination.

The first step in the operation is to make a long incision sufficient to allow the tumour to be turned out of the abdomen. As soon as it is removed, the intestines are protected, either by covering with a large flat sponge or by putting temporary sutures through the abdominal wall, and catching them together by pressure forceps. The next step is to secure the broad ligaments, and tie the ovarian and uterine arteries. The success of this depends on the arrangement of the vessels of the uterus. You will remember that the uterine supply is obtained from both ovarian and uterine arteries, that these unite by a branch which runs along the side of the uterus, from which smaller branches enter the uterine wall. The result of that is that the main arteries are outside the wall of the uterus, and can be tied without piercing the uterine tissue. In the case of a large uterine tumour, it will generally be found that the broad ligaments are very much stretched upward. The ovarian artery enters near the border of the broad ligament, with, in most cases, huge veins, while the uterine artery approaches the uterus near the base of the broad ligament.

You will also remember the relation of the ureter to the uterine artery. It lies about $\frac{1}{4}$ inch from the side of the cervix, and crosses a little below the uterine artery. It is not necessary to tie the ureter. I show you here the instrument I use for tying these vessels, a semi-blunt pedicle needle, with a curve of nearly 90°.

Now I will describe the way in which I carry out the operation, not intending to give the details to exactly correspond with those of the author of the method.

A double ligature of silk, Chinese twist No. 4, is passed through the broad ligament, between the ovarian and uterine vessels, at about the level of the internal os. One loop of this again transfixes, close to the border of that outer part of the broad ligament called the infundibulo-pelvic ligament, just below the fimbriated end of the Fallopian tube. This loop is tied first, and then the second loop, which includes the free edge of the ligament, and lies in the groove formed by the first. The object of that is to permit of cutting very close to this ligature without any chance of its slipping off. Then a pair of large pressure forceps are put on

above, to check the bleeding from the uterine side, and the broad ligament is cut through as far as the ligatures have been placed. Tension is relieved very considerably in that way, and the tumour can then often be drawn up much more than is possible in the first instance. That is done on both sides.

Next, an incision is made from one side to another, merely through the peritoneum, joining the two points where the broad ligaments have been partially cut, and passing about half an inch above the utero-vesical reflection of peritoneum. With the fingers the peritoneum is stripped downwards from the uterus and broad ligaments, taking the bladder with it, carrying the bladder and ureter out of harm's way. That allows the finger guiding the needle to be pressed down close to the side of the neck of the uterus, almost as low as the vagina, between the bladder and the broad ligament. A double ligature of silk is thus passed through between the ureter and the cervix, beneath the uterine artery, which can be felt pulsating. One loop is again made to transfix the edge of the infundibulo-pelvic ligament a little outside the ovarian ligature. This is first tied, and then the second loop, which includes the free edge of the ligament, and lies in the groove formed by the first. Thus the second double ligature includes ovarian as well as uterine vessels; and the ovarian and uterine ligatures together constrict up the broad ligament into a pedicle of small size. The ends of the transfixing loops are left long till the end of the operation, and serve to draw the stump up into view. The peritoneum on the posterior side must next be divided in the same way as on the anterior side, and stripped down a little, so as to leave an edge of it to sew. Then the uterus is cut through transversely, a little above the level of the uterine ligatures, and below the level of the internal os. If the ovarian and uterine arteries have been properly secured you get very little bleeding from that surface. Some small vessels bleed occasionally, but in these cases I have found it sufficient to use pressure forceps for a few minutes, and not leave any ligatures at all. These vessels are supplied by a branch to cervix and vagina which comes off from the uterine artery outside the ureter, and generally escapes the mass ligature. The next step, after disinfecting the cervical canal with perchloride of mercury, is to sew together the peritoneum in front and at the back by a continu-

ous suture of thin chromicized gut. This I have carried out, not by the ordinary method of piercing the peritoneum twice on each side, but by adopting the principle advocated by Mr. Greig Smith, i.e. to secure a sero-fibrous union. The suture pierces the peritoneum twice on one side and once on the other side, so that a flat surface of peritoneum is brought against a divided surface of cellular tissue. The principal reason of that is that peritoneum brought to peritoneum unites rapidly, but is easily separable. A purely fibrous union is stronger, but slow. Cellular tissue with peritoneum effects an union which combines the advantages of the other two—the union is quick and firm.

In this way the stump of the uterus is rendered extra-peritoneal; the pedicles formed by the broad ligaments are left intra-peritoneal, and are generally not larger than those formed in a double oophorectomy. A kind of pocket is formed between the peritoneum and the cut surface of the uterus, and the dexterity of the operator is shown in so adjusting the lengths of peritoneum left as to make the pocket as small as possible; because there must be a certain amount of sanguineous oozing into it. The blood so effused can drain, in some measure, through the cervical canal. Some recommend the turning of the ligatures of the broad ligaments, including the uterine and ovarian arteries, into the same pouch. This can be done by uniting the peritoneum over the ligatures, making them extra-peritoneal. But I think that is undesirable, and I adopt the plan of leaving absolutely no ligature or suture in the shut-off pouch, letting the ligatures be intra-peritoneal.

Baer's operation has probably not been performed often enough in England to allow of a complete estimate of its mortality. American statistics give a mortality of about 5 per cent., and this is probably capable of improvement. It compares favourably with that of other modes of hysterectomy. But one may judge something of the danger of an operation by the mode in which patients recover. So far, all the patients on whom I have operated by this method have recovered as rapidly, and with as little cause for anxiety, as after an easy ovariectomy.

This is a contrast to our experience in supra-vaginal hysterectomy, with external treatment of

the pedicle. I show you here the instruments used in that case. The stump is surrounded by a wire attached to a *serre-nœud*, fixed in the abdominal wall by means of pins, which draw it up, and keep it on a level with the surface; and the peritoneum is closely adapted round the stump. I have myself adhered to that operation for a long time, finding that it gives excellent results as regards ultimate recovery, providing that a stump of moderate size can be obtained and brought to the surface without extreme tension. It, however, has serious disadvantages; convalescence is protracted, owing to the sloughing of the stump; there is often a good deal of constitutional disturbance during the process of sloughing. The patient is in bed for six weeks, possibly longer, and finally there is left, at the site of the stump, a weak spot in the abdominal wall, which will, in some cases, give rise to ventral hernia, even after the lapse of a considerable number of years. The intra-peritoneal treatment of the pedicle has therefore strong claims to preference, if its rate of mortality proves to be merely equally favourable.

In this method of hysterectomy, the only risk of ventral hernia arises from the considerable length of the incision. If care be taken in uniting that incision, the risk can be practically abolished. The mode in which the abdominal wall is united in such cases varies in the hands of different operators. It is usual to use three sets of sutures. I prefer to have one set of deep sutures of silkworm gut, going through everything, including the muscles and peritoneum. Then I apply a continuous buried suture of chromicized gut; and this is the point on which I specially rely in preventing risk of hernia. It is used to unite the edges of that part of the transversalis fascia which is superficial to the recti muscles. This is really the only structure in the course of the abdominal incision which affords any firm support, being attached to the transversalis muscles at the side. It is better, I think, not to take up much more tissue than the transversalis fascia itself, in order to more absolutely secure that the fascia is closely united. In case the recti muscles seem inclined to separate, I include some fibres of the muscle in every second or third stitch. Finally the skin is united by a third set of sutures of silkworm gut.

I will now give you short accounts of the cases which illustrate the points I have put before you.

Case 1.—J. R., married nineteen years, 41 years of age; had no pregnancy; husband living. She was admitted September 14th, 1895, on account of abdominal tumour. Menstruation had been regular, occurring once a month, lasting three or four days and not excessive in amount, nor did the patient pass clots; therefore the operation here was not indicated by menorrhagia. She had very little pain. Twelve months ago the patient noticed the right side of her abdomen getting hard, but a lump was only definitely recognized three months ago. The last period was a week before operation. There was difficulty in micturition twelve months ago. On examination, a large mass was felt, reaching above the umbilicus, highest on right side. Vaginal examination showed that the cervix uteri was pushed forward close to the pubes, and that a large, firm mass filled the whole of the pelvis behind it. It was continuous with the tumour felt externally. The diagnosis was that of fibroid tumour, and the indications here were that the tumour was situated in the pelvis, and was therefore likely to interfere with the ureter or bladder if it grew much more, particularly as there had been previous difficulty in micturition. She was also anxious for relief and complete restoration to health, having to earn her living. Operation was therefore decided on, and was performed on September 30th by the method I have described. The only difficulty which arose in the operation was that on turning the tumour out of the abdomen, it was found that the mass which was felt in the pouch of Douglas, although it was possible to turn it out, had descended quite low, involving part of the cervix. Therefore it was difficult to reduce the pedicle to a small mass consisting of the cervix only. Accordingly, when the peritoneum was united by sutures, the pocket formed was of considerable size. The peritoneum had not been shortened quite sufficiently, and when the continuous suture had been nearly completed it was found that a considerable amount of bleeding was taking place into the pocket. Some interrupted sutures were put in more deeply, including part of the wall of the bladder and the deeper margin of the posterior peritoneum. The operation was completed in the way already described. The only unfavourable symptom which was manifested afterwards was that on the day after operation the urine was found to contain

a good deal of blood, and albumen due to the blood. It was thick, and of the colour of port wine. The temperature and pulse were moderate, the temperature being highest on the evening of the operation, viz. $100^{\circ}4'$. From the condition of the urine, it seems clear that these subsequent stitches, or one of them, put in to arrest the bleeding, must have pricked the bladder, but no serious harm resulted from it.

On the 3rd, viz. three days after the operation, the temperature had fallen to 99° , and the urine was free from albumen and blood. The bowels acted on the fourth day, and a fish diet was ordered the day after. The deep sutures were removed on the seventh day, and the remainder two days later.

On the 18th (eighteen days after operation), the patient got up; so that the convalescence was extremely rapid.

The next case was that of I. F., aged 43, admitted October 14th, 1895. She had had one child, which was born in March, 1875, she having been married twenty-two years ago. She had had one miscarriage, and had married for the second time seven months ago. Menstruation occurred regularly every fourth week until two years ago, when the menses came on every three weeks, lasting four days. The last period was in June, after which she saw nothing until August, when it lasted three weeks. There had been no recurrence since August 21st. She was well until June, at which time she noticed she was getting larger, and attributed it to pregnancy. In August the discharge was of a dark brown colour, and when applying at the hospital she complained of great weakness, a feeling of distension in the abdomen, and a smarting pain in the groin.

On examination, a large swelling was felt in the lower part of the abdomen. The tumour evidently consisted of two parts, the left being firmer and giving a direct impulse to the cervix, and a very loud uterine souffle. This was thought to be the pregnant uterus. The right-hand portion was larger than the left, was softer, and gave a feeling of fluctuation and a slight fluid thrill. The great point to settle was whether the tumour was ovarian or uterine. On some occasions a very slight souffle could be heard over the tumour, but at others none at all.

The question was discussed whether this faint

sound proved the tumour to be uterine, or whether it might merely be transmitted from the other side of the abdomen, where so loud a souffle was heard. On the whole, I was inclined to think the tumour more likely to be ovarian; but, in either case, it was so placed at the brim as to be likely to obstruct labour, so it was decided to operate, especially as the tumour was increasing rapidly. (It trebled in three weeks.) The operation was performed on October 21st, in very much the same way as in the other case, and there was even less trouble than I have usually found in securing the vessels. The pregnancy did not seem to complicate that part of the operation to any serious extent, although the vessels were large and the veins enormously distended. It proved to be an oedematous fibroid, though it does not now look different from any ordinary fibroid, the fluid having oozed out of it, and the part which contained the foetus has contracted up to a great extent. On opening it was found that the placenta, although not over the internal os uteri, was pretty low down in the direction of it, so that it very likely might have appeared as a marginal placenta praevia when the labour came on. The foetus corresponded to four and a half months' pregnancy. It should also be noticed that the tumour extended in the wall of the uterus down to the level of the internal os uteri; so that the probability is, if pregnancy had been allowed to go on, it would have been an obstruction to labour.

The patient recovered with as little disturbance as the other one. There was the usual discharge from the vagina, the maximum temperature was 99.8°, and the urine remained normal. The deep stitches were removed on the eighth day, and the superficial ones two days afterwards, and the patient was practically convalescent at the end of a fortnight.

The third case was that of C. S., single, aged 39, admitted September 23rd, 1895, and I have the tumour here to show you. There was one very large fibroid in the wall of the uterus, and other superficial fibroids of a sub-peritoneal character. This was another patient who had to earn her living and was anxious for relief. The tumour reached to an inch above the umbilicus, and was most prominent on the left side. There was a large mass in the pouch of Douglas, behind the uterus, which, as in the first patient, would be likely to lead to inter-

ference with the bladder and ureter. The operation was performed October 25th. There was considerable difficulty in delivering the tumour through the abdominal incision, and in doing so the peritoneum, on the posterior wall of it, tore across rather low down, near the bottom of the pouch of Douglas. That tear was utilized as the posterior division of the peritoneum, and it was found possible to stitch the peritoneum over the bladder to it.

In this case there was a little more trouble than in the others due to bleeding. In passing the needle on the right-hand side to tie the uterine artery, apparently a vein was transfixed, therefore it was necessary to carry a second loop somewhat deeper down, and closer to the cervix uteri, in order to secure all the veins, although the first ligature had successfully surrounded the uterine artery. The only noticeable item in this patient's convalescence was that on the night following the operation there was rather more vaginal haemorrhage than in the other two patients, coming, no doubt, through the cervix uteri. Some blood clots were passed during that night, but the patient remained free from harm, and is now convalescent. The rise in temperature was very slight indeed, and the pulse continued practically normal.

A particular case in which this method of carrying out the operation of hysterectomy might not answer would be that in which the fibroid tumour descended so low into the cervix as to involve the vaginal portion. In that instance I believe it would be better to adopt the plan of total hysterectomy, removing the cervix also.

Another case in which the difficulties are very considerable is when there is an intra-ligamentous fibroid. In that case it may be necessary to enucleate the fibroid from the broad ligament before tying the uterine arteries. If the gap so left be moderate, it may be closed up by deep and superficial sutures of chromicized gut, and the operation completed according to Baer's method. If it is very large, or if the bleeding into it is not easily controlled, it is, I believe, better to perform total hysterectomy, removing the cervix also. The cavity is packed with iodoform gauze, which is carried out into the vagina so as to form a drain. The peritoneum is united by a continuous suture of fine gut, in such a way as to make the pedicles and ligatures extra-peritoneal. The ends of the ligatures are left long; and, being passed down through the vagina and out at the vulva, serve to draw down the pedicles formed by the broad ligaments towards the vagina. The pedicles ultimately slough and come away with the ligatures. This is the method advocated by Martin, of Berlin, for all cases. His statistics give a mortality rather under 7 per cent.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 18, 1895.

A CLINICAL LECTURE

ON

MYELITIS.

Delivered at the Bristol Royal Infirmary,

By HENRY WALDO, M.D., M.R.C.P.,

Physician to the Infirmary.

GENTLEMEN,—There is a young man in one of my beds with partial loss of power in the lower part of his body and legs. His sensation, too, is somewhat interfered with. The notes of the case are the following:—He is a china-ware hawker, aged thirty, single. He applied for admission owing to weakness of his legs. His father died at thirty of heart disease. Mother alive and well, aged fifty-eight. No family diseases. Has always taken alcohol in moderation. Usual health most robust. He contracted a primary syphilitic sore three years ago, for which he applied to a chemist. This was followed in a few weeks by a skin rash all over his body. For this he was treated at the Stroud Hospital for two months. Eighteen months after the primary sore, having remained in his wet clothes for some hours, he felt chilly, and found that he could not pass urine. Three weeks after this he noticed tingling and heaviness in his left leg, which was quickly followed by some loss of power. These symptoms extended to the right leg, but not for three or four months. He had sharp pains in the legs during the night only. Soon after the left leg was affected, he had a "girdle pain." About this time he suffered with headache and giddiness so much that one day he fell down. He also vomited. The temperature since admission has been normal. There have been no rigors. The cardiac sounds are clear. Pulse eighty, regular. Ruddy complexion. Respiration natural. No cough. No headache or giddiness. Rather much tenderness of spine from the sixth dorsal to the second lumbar inclusive. Pain also here which has prevented his sleeping well for the last twelve months. Walking about has always increased

this pain. A warm test tube passed down the back increases the pain at this site. There is a band of hyperæsthesia two inches below the umbilicus, narrower on the left than the right side. Partial loss of power in both legs. Only slight wasting. He says the legs feel numb. There is some anæsthesia, especially to heat. Prick of pin is not felt much over both limbs, especially below knees. Sensation to temperature is nearly absent below the right knee. Painful impressions are felt at once, and there is no loss of muscular sense. He complains of his feet and legs "going very cold and then burning like a fiery coal," and also of occasional aching pains in his knees. He can walk with assistance, his gait being somewhat spastic. He says the floor feels harder than formerly. He has used crutches for some time. There has been no sign of a bedsore. The superficial reflexes are all absent, with the exception of the plantar, which is normal. The deep reflexes are much exaggerated. His pupils are unequal, the right being the larger; both act to light and to accommodation. Tongue fairly clean. Bowels loose and open several times daily since admission. Before this they were very constipated. His appetite has always been good.

I believe this patient's symptoms depend upon a syphilitic affection of the spinal cord in the dorsal region. As we go on, I will give you my reasons for making this diagnosis. His occupation necessitated his being much exposed to the weather when his body was heated. Cold, especially wet cold, is undoubtedly an exciting cause of myelitis. It acts by producing a depressed surface temperature, and so suppressing the functions of the skin. Myelitis has been produced in animals by varnishing their skins. Lying upon damp cold ground or bathing in very cold water when exhausted has been thought to act in the same way. Over-exertion is another exciting cause which I think played its part in this case, for the patient says he was often very tired. Sexual excess is also considered as tending in the same way, but there is no evidence of this here. No railway accident or other injury had

occurred to this man. Another cause—a predisposing cause—and one which was more important than any other in producing the symptoms, was the poison of syphilis. The nervous system is often selected in syphilis, and sometimes in a very early stage, but it is very exceptional for any disease of the nervous system to occur earlier than the sixth month, and as a rule it is a late affection. In syphilitic myelitis, it is nearly always the dorsal portion of the cord which is attacked, as in our case. Eighteen months after the primary sore, the cord symptoms commenced with difficulty in passing urine. I have known this to be the first symptom noticed in other cases of myelitis. Owing to the volitional path in the cord being interrupted above the lumbar centres, the detrusor muscular fibres of the bladder, as well as the abdominal muscles, refused to contract. This condition soon passed off, to leave reflex incontinence, as the result of the motor centre in the lumbar bulb yielding too readily to the afferent impulse. Since being in the Infirmary he has much improved, and all that he complains of now in this respect is that he has to wait for some little time before he can urinate.

The next thing to attract this patient's notice was perverted sensations, or what are called paræsthesiæ—in this case consisting of numbness, a feeling of "pins and needles," and heaviness—in his left leg. There were also some sharp pains in his legs. But there has been an absence of radiating or extrinsic pain around the trunk, and this is an important point in excluding caries of the vertebræ and also tumour, in which you get more distinct pressure signs. As regards caries, this young man has no tubercular family history, which goes for something; and upon examining the spines of his vertebræ, there is no displacement. Again, although there was a good deal of pain in the back, it was not increased by twisting the spine. Where there is the least reason to suspect caries, do not fail to constantly examine the vertebral spines, and pay attention also to any radiating pain, as caries has been mistaken for intercostal neuralgia, stomach-ache, and even renal colic—as you can see described in Hilton's work of "Rest and Pain." There is distinct tenderness from the sixth dorsal to the second lumbar vertebræ. You may get this in myelitis or caries, but as a rule it is a rare symptom in tumour. A girdle

pain sometimes occurs in cases of myelitis, and is an indication of interference with the posterior part of the cord. It much oftener depends upon pressure, and so is more frequently present in caries or tumour. The zone of hyperæsthesia is the result of irritation at the junction of the upper end of the inflamed and the healthy cord, and especially of the nerve-roots. It should be frequently watched, as it only shifts its position with the myelitis. In this case it is below the centres for the intercostal muscles, and they are not affected. The sensory symptoms in the legs and feet are very slight as compared with the loss of power. Gowers has quite recently said that "It is a rule with the nerves and cord alike, that an apparently equal affection of all the structures has far more effect on the motor structures than on the sensory." You may rely upon it that if sensation is much interfered with, it is a severe case. The loss of power in the legs was not sudden but gradual, and so you can exclude hæmorrhage as the primary cause. An unusually long period is said to have elapsed between the weakening of the left and right leg, but there is no evidence of a crossed paralysis of motion and sensation. The motor paralysis was never complete; it was more a paresis or partial paraplegia. This patient is now in a condition of more or less spastic paraplegia, although there is very little rigidity present. Spasticity is the clinical disclosure of degeneration in the motor tract, and the cause of the degeneration is that the dorsal lesion has cut off the intra-spinal motor fibres from the nerve cells in the brain cortex, from which they derive their vitality. The spastic symptoms are illustrated by a rectus clonus on each side, excess of knee jerks, Gowers' front tap contraction, and ankle clonus. These signs show you that the lumbar enlargement is not involved, as if it were, all reflex action would be lost and the leg muscles would be flaccid and waste, besides giving the reaction of degeneration to electric currents. The electrical reactions in this case are normal. The urine, too, would escape from the bladder as soon as it got into it if the lumbar bulb is affected, and there would be incontinence of fæces. And in addition to this, there would be bedsores over the sacrum, a tendency to which is rarely great unless the lumbar enlargement is compressed or myelitis descends into it. The superficial reflexes are all absent, except the plantar,

which is normal. This fact points to a myelitis rather than to caries or tumour. Gowers says: "Excess of superficial reflex action is a common symptom in cases of compression of the cord, as distinguished from damage to the cord at a similar level by a primary morbid process within it."

The pathology of what is called syphilitic myelitis is, at any rate in some cases, more a softening of the nervous matter than an inflammation of it. As in the brain so in the cord an arteritis may be set up. The lumen of the vessels implicated lessens until they contain blood clot. This thrombosis cuts off the blood supply, and as a consequence the part softens. Probably this is what occurred to some of the brain arteries in the early stage of this man's illness, when he felt giddy and fell down. A similar affection of the vessels in the spinal pia mater would explain the cord symptoms. But you must remember that it is impossible to draw a distinction between cases of transverse myelitis, probably syphilitic, and similar cases in which syphilis can with certainty be excluded; and that paraplegia may be due to a syphilitic myelitis and not as a result of arterial disease.

In the discussion at the Royal Medical and Chirurgical Society in the early part of this year, on the affections of the nervous system occurring in the early (secondary) stages of syphilis, Mr. Jonathan Hutchinson showed some illustrations of the microscopic appearances of the spinal cord in acute paraplegia occurring in the early stage of syphilis, which he had had copied from an account of a case published by M. Lamy, of Paris. "The patient was a man aged fifty, admitted with complete paraplegia affecting motion, sensation, and the sphincters. He had contracted syphilis one year before, and when seen presented a syphilitic eruption. The paraplegia lasted two weeks, the patient developed a large bedsore, and died suddenly. At the necropsy the spinal cord and membranes were normal to the naked eye. There was no softening, so that the early minute changes could be clearly seen, and were not obscured in any way. The stress of the disease had fallen on the perivascular spaces of the vessels, especially the veins, and had respected the nervous elements. In the grey matter the perivascular spaces contained colloid matter; in places the veins were thrombosed and showed microscopic gummata in

connection with them. There was some degeneration around the septa running in from the pia mater, but most of the nerve elements were normal. These changes were present throughout the whole of the cord." The inequality of pupils may be owing to a lesion in the cervical region of the cord, as syphilitic myelitis may be of the disseminated variety. But I think this interpretation of the unequal pupils unlikely in the absence of other signs.

The prognosis in this case is fairly good, and this is principally owing to the dorsal portion of the cord being alone affected. It would have been more favourable if efficient treatment had been commenced earlier. Inflammation of the cervical cord would be attended with much more serious symptoms, as the respiratory muscles may be interfered with, but up to the present there is no sign of the disease ascending. Another good point is that the lumbar enlargement is not involved, and so there is little likelihood of the bladder and kidneys becoming inflamed and ulcerated. When pyelo-nephritis occurs, a fatal termination may be predicted within three years. Yet another favourable sign is that sensation is very little interrupted. As regards the spastic state of the legs I expect very little amelioration on account of the primary change having been a myelitis. Had this been pressure there would have been good hope of at any rate some recovery. In a clinical lecture given by Dr. Gowers a few weeks ago, he says, "The spastic state is capable of passing away completely if the pressure on the cord is removed. There is no degree of spastic paralysis that may not be recovered from if its cause can be removed before there is total destruction."

The treatment adopted has been rest in bed, a liberal general diet, counter-irritation with linimentum hydrargyri, gradually increasing doses of the iodides, and cod liver oil. If the lumbar bulb is affected it is better to use a water bed from the first, or should you suspect a bedsore, whether the lumbar bulb is affected or not, you had better use one. I usually have it filled partly with warm water and partly with air. It is important to attend to the temperature of the water, especially in cold weather, by having it changed occasionally, or the patient may get chilled. In treating these cases with anti-syphilitic drugs you must not expect too

much. If the vessels are thrombosed it is considered very doubtful whether they ever become pervious again; the majority of observers believe they are permanently converted into fibrous cords. But specific treatment, if begun early, can prevent other vessels from following in the same train. The nerve lesions of syphilis seem to be more amenable to the iodides than to mercury, but there are some exceptions to this. Whether you use these drugs separately or in combination do not keep up this treatment for longer than six or eight weeks at a time, but give the patient a rest between with, perhaps other drugs that may be necessary, as iron or quinine. When giving iodides gradually increase the dose from, say, five grains up to thirty, three times a day. In using mercury never give enough to salivate, as if this should arise it quite prevents your going on with the treatment for a month or two. I find a thirty-second of a grain of the bichloride, given twice or three times a day, quite sufficient, and there is then no fear of salivation. Mr. Hutchinson says that early affections of the nervous system in syphilis occur almost invariably to those in whom the treatment in the early stages of the disease has been more or less neglected. He does not dispute that at much later periods in the course of syphilis affections closely similar to these may occur, but he believes that when they do so they are much slower in onset, less severe and more chronic in progress, and less amenable to specific treatment. Sir W. Broadbent thinks that the therapeutic effects of mercury and iodide of potassium would ultimately be explained by their effects on the toxic bodies giving rise to the secondary and tertiary lesions. Dr. Gowers, arguing on the analogy of the diphtheritic organism, is inclined to think that tabes dorsalis (locomotor ataxy) is due, like diphtheritic paralysis, which sometimes closely resembles it, to some chemical result of the syphilitic organism; while Hutchinson thinks the tertiary lesions of syphilis are not due to the action of toxins, but to local degenerations in the cells. It may be necessary to relieve pain with morphia given hypodermically, but the pain in these cases of syphilitic myelitis quickly responds to specific treatment. Soon after the linimentum hydrargyri was rubbed into this man's back he lost the pain which had prevented his sleeping so long; it no doubt acted partly by irritating the surface and partly from absorption of

some of the metal. A sea voyage is considered to be most beneficial for these patients, as they get the advantage of much fresh air with little exertion.

CLINICAL DEMONSTRATIONS

AT THE

CENTRAL LONDON SICK ASYLUM,

Cleveland Street, November 21st, 1895.

By THOMAS BRYANT, M.Ch., F.R.C.S. Eng. & Ire.

Consulting Surgeon to Guy's Hospital.

LADIES AND GENTLEMEN,—Three cases have been selected for our consideration to-day, and it so happens that all of them illustrate some affections of the circulatory system, or, more correctly, affections connected with the blood channels. The first to which I will draw your attention is that of aneurysm of the arch of the aorta.

The patient is a childless widow, fifty-nine years of age; when forty-five years of age she had rheumatic fever, and has since then been treated for heart trouble, neuralgia, and rheumatism at several hospitals. She has also had palpitation for five years. A swelling in the neck was noticed on the right side, above the sterno-clavicular joint, about two years ago, and severe neuralgia has troubled her for the last eighteen months; besides which she has had many attacks of dyspnoea. These attacks began with pain about the swelling which is so palpable over the right sterno-clavicular joint, and passed up to the head on the right side, and down the right arm. At times she has great pain in the right parietal region and about the ear, accompanied by a feeling of faintness and distressing breathing. The report says the patient is aware that at times the breathing ceases entirely, and at the same time she loses the power of speech, but remains quite conscious. She has had two such attacks since her admission into this institution, and at these times, when seen by the nurse, she appeared pale, complained of faintness, and was breathing quickly. The attacks of dyspnoea were broken by periods of complete

apnoea, the patient lying absolutely motionless, with eyes closed, for a period of about fifty seconds; then dyspnoea began again, the patient became restless, and appealed for something to be done to relieve her. Relief was then quickly obtained from nitrate of amyl. The heart is hypertrophied, and there is mitral disease. The vessels at the bend of the elbow and at the wrist are hard and tortuous; there is no decided inequality of the pulse at the wrist. A pulsating tumour exists behind the right sterno-clavicular articulation and the upper end of the sternum, projecting upwards and backwards into the neck. There is evidence, also, in some of her joints, of arthritis deformans.

There can be no question that this poor woman has an aneurysm of the aorta. You will see that it causes a great pulsating swelling, which passes above the right clavicle and an inch above its articulation with the sternum, suggesting a probable implication of the innominate artery as well. In support of that view we would draw your attention to the extreme pain which shoots down the arm, and the general wasting of the limb, these facts suggesting that the aneurysm is pressing upon the brachial plexus. My friend Mr. Hopkins tells me that he is not able to make out that the pain follows definitely any one nerve trunk, but it seems to include all.

This question of apnoea and dyspnoea is a very interesting one, and possibly it is somewhat difficult to explain. Apnoea unquestionably at first raises the suggestion that there must be some nerve central cause for it, and this view, of course, opens up a wide field for speculation. As we recognize there is general arterial disease, it is reasonable to suspect that this condition is likely to have extended to the vertebral artery, with other cerebral vessels, and so involve the respiratory centre. But I am not going to give you that as an explanation. I might have been tempted to have done so had I not, during the last few months when looking over my notes of cases, encountered a record of a very exceptional case, in which apnoea and dyspnoea existed together, as in the instance before us, but where there was no such marked evidence of aneurysm as is presented by this patient. It occurred in a young married woman, aged only 22, and her apnoea and dyspnoea was said to have threatened death; indeed, she was sent to me at

Guy's Hospital with that history. She appeared, however, to be in perfect health, so much so that it was suggested she should not be kept in the hospital, and some of those present assumed—hastily assumed—that it was a case of hysteria, for on a most careful investigation of her case nothing wrong, that is, no objective symptom, could be made out. Now I am, personally, a firm believer in the value of a history of a case, and have no sympathy with those practitioners who have so much self-confidence in their own powers of observation that they ignore history, and only trust to and rely on what they see themselves. I regard this habit as a wrong and bad one, for by its adoption we often throw away our one chance of learning how to do what is right. Experience has convinced me that we must accept the histories of cases, although we may do so with the sceptical spirit which is allowable to a scientific man. This young woman looked well and was very intelligent, and when she heard that she was likely to be sent away she appealed to me, saying, "Don't let me go; if you do I shall surely die." I replied that she should not go, but that we would watch her. And it was well that I so acted, for on the night of the second day she was seized with dyspnoea, which threatened suffocation. The house surgeon was sent for, and he naturally thought of tracheotomizing her; indeed, as the symptoms became worse he proceeded to carry out that operation, but before he completed it apnoea came on and the patient died. I should have stated that it was noticed during life that this young woman had no pulsation whatever in the left arm from the shoulder downwards—no axillary, brachial, or radial pulsation, but that condition did not then appear to have any connection with her trouble. At the post-mortem, however, a sufficient explanation of her death as well as of the absence of pulsation of the vessels of her left arm was readily found by the existence of an aneurysm of the aorta, $1\frac{1}{2}$ inch below the orifice of the left subclavian artery. It was about the size of a plum, and so pressed upon the trachea and the left bronchus as to completely flatten them. But in addition to this aneurysm there was this curious fact—she had a completely occluded left subclavian artery, and the circulation of that limb seemed to have been carried on through the inferior thyroid artery. I give you this case as an interesting one, as it bears in a measure on the

one before us; indeed, the case I have briefly referred to completely tallies with the description which Mr. Hopkins has given of the dyspnoea and apnoea of his patient, and it is quite possible that in the case before us there may be some bulging of the aneurysm backwards as to press upon the right bronchus, and so involving it, give rise to her symptoms. But to proceed.

I will not go at any length into what could have given rise to this aneurysm, because you all recognize that aneurysms may be due to anything which tends to weaken the coats, and particularly the inner and middle coats, of an artery; the external coat we need not take much into account, for it is rarely the seat of disease, though injury from external agencies may so weaken it as to produce aneurysm. Doubtless there has been here some disease weakening the inner and middle arterial coats, and, following that weakening, expansion. The disease has in all probability been what we call atheroma. I might remark here that atheroma is not always recognized as it should be. Students often say it is the fatty degeneration of an artery. In a measure they are right in so saying; but they should bear in mind that a perfectly healthy artery will not undergo fatty degeneration, there must be something more to degenerate than healthy cells. Therefore there must be some previous deposit of something to degenerate, and pathologists now fully understand that atheroma is merely fatty degeneration of some former inflammatory products which have been poured out but not re-absorbed.

Atheroma is consequently the secondary result of an antecedent local arteritis; the inflammatory deposit between the inner and middle coats of the artery, instead of undergoing absorption and permitting the vessel to recover, or of undergoing a fibroid change and causing thickening of the walls of the vessel, takes on a degenerative action and becomes fatty, and thus forms an atheroma.

What, then, is the treatment for this affection? This patient seems comfortable, does not now seem to be in pain, takes her food kindly, and the machinery of life goes on fairly satisfactorily. When she does feel pain it is pressure pain, and the question arises, "Can't we do anything to help her?" You know, in such a case as this, that medical relief cannot do much, and the mind consequently passes to surgical treatment. I have been tempted in times past, where in cases of this

kind great pain has been present, to tie a distal artery, and in that way reduce, so to speak, the force of the blood current through the aneurysm, and thus give relief. I have done so in three separate instances of this kind, and in all of them relief was afforded almost immediately. From a consideration of the local conditions alone this line of treatment appears, therefore, reasonable. But in thus speaking we must not lose sight of the individual, the patient herself; and I do not think that the most confident or hopeful surgeon would in this patient consider such a measure to be either expedient or right; her feeble general condition forbids the act. Under the circumstances we can only adopt measures of relief and subdue pain as far as possible by the cautious use of sedatives.

You will remember I told you that there was no decided inequality of pulse at the wrists, and the reason of this I believe to be as follows. As a rule, when an aneurysm is present in a main artery of a limb, the circulation in that limb is interfered with, for the pulse wave must of necessity be considerably altered by the presence of an aneurysmal sac or an arterial dilatation in the continuity of the vessel, and as a consequence there would be a marked difference between the pulse of the affected and the unaffected extremity. In the case before us these conditions do not exist, for the aneurysm is in the arch of the aorta, and consequently the left side is affected in the same degree as the right. This view is an important one, because in the diagnosis of some cases it must help you. In subclavian aneurysm, for example, you will find a difference in the radial pulse of the two sides, whereas in aortic aneurysm this difference will not exist, since in it the circulation is affected generally, and although the pulses of the body may be affected by the aneurysm, there will be no difference in the character of the pulse on the two sides.

Case 2 will now engage our attention. It is one of senile gangrene in a male patient 75 years of age, and for which a Syme's amputation has been performed. There is too much confusion of thought on this subject of gangrene; I propose therefore to make a few remarks upon it as a whole. Clinically, I have always been disposed to divide the subject of gangrene into three main divisions.

First, gangrene due to cutting off the arterial supply from a part, and thus bringing about its death from starvation of tissue. No blood is sent to a part on account of some arterial obstruction, by such as that produced by what is known as embolism; by arterial thrombosis, the result of injury, this condition being equivalent to embolism, or by some local injury separating and dividing the inner and middle coats of an artery, and thus allowing them to recurve and block the lumen of the vessel in continuity in the same way as torsion is now known so successfully to arrest bleeding in a divided artery. The same result may also be brought about by the application of a ligature to the trunk of an artery by a surgical process. Under all these circumstances there is danger of gangrene, due to the cutting off of the supply of blood from a part. This variety of gangrene should be called anæmic gangrene—gangrene from want of blood supply. The second form of gangrene is due to a totally different cause, that of venous obstruction, the obstruction to the return of blood from a part. This is seen in practice, in cases of strangulated hernia and strangulated piles, as well as when in cases of fracture, splints have been too firmly applied. The gangrene is due, under these conditions, to blood stasis in a part, and should be called static gangrene, to distinguish it from the former group. The *third* variety of gangrene should be called septic inflammatory gangrene—gangrene due to the inflammation of a part. The second and third varieties of gangrene are, as a rule, moist. The first is, as a rule, dry gangrene, unless the case, in its progress, becomes complicated by some venous obstruction, or some inflammatory action. The division of gangrene into dry and moist is not, however, a good one. An inflammatory gangrene is nearly always a septic and likewise a spreading gangrene, by the infiltration of the connective tissue of the affected part by septic exudation; these exudations infecting the tissues mechanically by following the lines of least resistance between the muscles and beneath the skin. The gangrene due to "frost-bite" is not due to either arterial occlusion or to venous obstruction, but to the direct effect of cold upon the tissues, by which the blood is expelled from and not allowed to re-enter the part; and when gangrene follows as a direct result of cold, it may be described as an example of anæmic gangrene. If the case is not treated judiciously and well, other conditions

arise, for if a reaction in the frozen tissues is brought about too rapidly, and relaxation of these tissues takes place, blood is sent into these paralyzed and relaxed capillaries, when the power of forcing it through the tissues into the return veins no longer exists, and as a result static gangrene is brought about.

As to senile gangrene, the ailment of the patient we are now considering, it is of a mixed nature. It occurs in either old persons, or in young persons prematurely old, with diseased arteries, and is very seldom brought on by what I may term purely natural processes; there is generally some traumatism mixed with it. But let us return to our case, the report of which tells us that he has always been temperate, and has had three grown-up children, and has never had syphilis. When his trouble first appeared, he suddenly felt a pain in the big toe when getting out of bed, which prevented him walking. The toe then rapidly turned black, and the remainder of the toes soon followed suit and became similarly affected. For this trouble amputation at the ankle-joint was subsequently performed as a Syme's amputation, and you now see a splendid stump.

The sudden pain the man felt on the onset of the affection was probably due to a sudden cutting off of the blood supply to the part; we may call it a cry of pain from the affected part from want of food. A patient with sudden embolic plugging of any artery will very commonly cry out from sudden pain, as if he had been struck in the part. A likely cause of that sudden stoppage of the blood supply is that the patient has atheromatous vessels, causing roughness of some part of an artery in the lower extremity, and that the blood in passing over this roughened surface has been whipped of its fibrin, and that a small clot there formed; that this clot, either alone, or associated with fatty, atheromatous material, was carried from the place at which it was formed down to a vessel through which it could not pass, and that as a consequence an embolic plugging took place.

This sudden blockage with small fragments of fibrin or of atheromatous or calcareous deposit is generally the beginning of senile gangrene, unless the element of traumatism has been introduced, in the form of some trivial injury, such as that produced by the cutting of a corn. A local injury in a part badly supplied with blood from being

carried through diseased vessels being followed by a local inflammation which goes on to gangrene; the blood stasis which is always present in an inflamed part bringing it about.

In this case there is no history of traumatism, so that we are free to assume that it was entirely due to obstructed vessels, and the history well supports that view, for it spread to other toes and involved them, and travelled up as far as the metatarsal bones, the heads of some of which were exposed. Therefore the surgeon at this stage of the process wisely amputated the foot, and from the excellence of the stump you may be sure that the operation was well performed.

You may ask why the whole foot was taken off, seeing that only the toes were involved. The answer to this question is, that the surgeon knew if he performed the lower operation in a case of this kind, in which the arteries were in all probability blocked, there would have been more sloughing for want of arterial supply; and in support of this view there is now no perceptible pulsation in the dorsal artery of the foot: the heart is weak and intermittent, and the patient is 75 years of age. The surgeon ran a risk in amputating where he did, but he was perfectly justified, indeed, bound to take the risk. The peculiar glazed condition of the toes on the foot which remains shows clearly that the part is badly supplied with nerve force as well as blood supply; so that the surgeon is to be congratulated on his success.

The few minutes of our time which remain will allow me to call your attention to a case of frost-bite, which took place in a woman 53 years of age.

The report of her case states that she has had no serious illness previous to the onset of her present trouble, and at that time she felt well. Three weeks before her admission in January last she got wet while going to work, through her boots letting in the water. The same night there was a severe frost, and the feet became numb and discoloured. She went to King's College Hospital, and remained there two weeks, subsequently being sent here. When admitted, the toes were black on the right foot, and some of the toes of the left foot were also affected at the tips. On July 24th, six months after the onset, the toes separated, and the condition of the feet you now see—the feet being minus their toes. What you

see is almost entirely the result of Nature's work; for the surgeon has, rightly, been content to look on while Nature has separated the dead parts from the living, being content to keep the tissues as clean and aseptic as possible.

Now, how can frost-bite be explained? This healthy woman got wet feet, and as the weather was bitterly cold in January, her toes became frozen. The toes did not go black at first, but were white, and perhaps a little mottled; the whiteness indicating complete anæmia, the mottling, capillary blood stasis. The blackness only came on as the blood entered the vessels, and the tissues gradually withered up. The case was one of arterial, venous, and capillary obstruction; then there was a little reaction, and all the other toes became involved, the disease spreading until at last the toes separated by the process of ulceration. Gangrene as a result of frost-bite is, if immediate, the death of tissue from anæmia, it is in fact an example of anæmic gangrene; but when this danger has passed and the case is not well treated, gangrene may still ensue, and it will then be an example of static gangrene from capillary blood stasis, more or less mixed up with septic inflammatory changes. It is upon a knowledge and appreciation of these dangers of both varieties of gangrene that the basis of the treatment of this class of cases rests. You are not to apply external warmth in the earliest stage of this affection under any circumstances whatever, because warmth would draw blood into the capillaries, which blood would stay there, and so add to the degree of blood stasis as to make matters worse. The old practice of rubbing the part with snow or ice is the correct one, the cold causing contraction of the tissues above the frozen part, and friction helping the circulation through them. The limb at the same time should be slightly elevated. I have seen many cases of frost-bite go on to the good under such treatment when persevered in; but the slightest neglect, half an hour's inattention perhaps, will make all the difference between success and failure. Never encourage any sudden flow of blood to the frozen part.

Much more remains to be said about these cases, but I believe I have pointed out the chief points of interest in each, and now leave the lessons which are to be learnt by their consideration for your serious thought.

A POST-GRADUATE LECTURE ON UTERINE HÆMORRHAGE.

Delivered at Charing Cross Hospital on November 21st,
1895,

By AMAND ROUTH, M.D., M.R.C.P.,

Obstetric Physician, with care of Out-Patients, and Lecturer
on Practical Obstetrics and Gynæcology to the Hospital.

LADIES AND GENTLEMEN,—The subject which I have chosen to-day needs no apology; uterine hæmorrhage is a trouble we constantly encounter. My object is to deal partly with the causes, and then with the appropriate treatment, so far as we can get it into a reasonable period of time; and I think it wise to go about it in the way we should adopt, if we had cases illustrative of the different forms before us. Thus, partly by the process of exclusion, we endeavour to arrive at the real origin of the hæmorrhage, because the great difficulty in dealing with uterine hæmorrhage is the diagnosis as to its origin; once this is ascertained, treatment is comparatively simple, at all events, we know what should be done.

To facilitate matters I have drawn up the scheme which I show you. First, *Hæmorrhages in connection with pregnancy*; secondly, *Hæmorrhages apart from pregnancy*. These practically include every variety of hæmorrhage.

In discussing hæmorrhages connected with pregnancy let us first take those *directly due to the existing pregnancy*, then those whose causes are not due to the pregnancy but *concurrent* with it, such, for instance, as carcinoma of the cervix complicating pregnancy.

I. *Hæmorrhages due to pregnancy*.—As a rule, we ought to be on our guard against the patient being pregnant in all cases of hæmorrhage. Undoubtedly many mistakes are made through not remembering the fact that a pregnant woman does not necessarily have amenorrhœa. It is very possible for a woman to be pregnant several months and yet to be losing blood; so that one would rather go to the opposite extreme, and say if a married woman comes complaining of metrorrhagia—irregular hæmorrhages every day or every

two or three days—and there is doubt whether she has missed a week or not, assume, for the time being, that she is pregnant, until you can exclude it, because the hæmorrhage is so often dependent on or concurrent with the pregnancy in one way or another.

The first group in hæmorrhages due to pregnancy are *threatened abortions*, or labours. Cases of *missed abortion* are not so easily recognized. A woman may have missed a period or two, followed perhaps by a sharp flooding, and then may begin to lose a little every day, or once a week, believing, excusably enough, that she is not pregnant. It is not uncommon to find cases of that sort, where, after a time, the ovum comes away, an ovum which, if it has been long enough in the uterus, may be considerably altered, becoming an apoplectic ovum or fleshy mole. The diagnosis of missed abortion is made chiefly by the fact that a period or two has been missed, followed by irregular hæmorrhages, and that the uterus is not increasing at the rate it should, yet is evidently too large to be merely a case of subinvolution.

Then there is *incomplete abortion*; bits left behind; or the fœtus may have come away, the placenta being left more or less entire. That, again, gives rise to metrorrhagic attacks, with possibly the absence of one period, but not always even that.

The next variety is *extra-uterine gestation*; one or two periods are missed, sometimes only one, then irregular hæmorrhages. Of course nothing but physical examination will enable you to decide that, in addition to the sympathetically enlarged uterus, there is also a tumour on one or other side, or dropping down into Douglas' pouch behind.

Then there are varieties of *moles*. Those usually met with are "blood moles" (apoplectic ova), which are due to hæmorrhages in the decidua or between the decidual layers; or a secondary "fleshy mole," which is generally due to hæmorrhages which have become organized, or complicated with a good deal of decidual endometritis, so that the whole structure becomes fleshy and much larger than the ordinary blood mole. They may remain *in utero* for months, usually acting as foreign bodies, and keeping up almost daily hæmorrhages. Sometimes the uterus becomes accustomed to their presence, and menstruation may for a time become regular again, but sooner or later the mole will be

expelled with smart hæmorrhage and some pain, which latter, however, may be occasionally almost absent.

Then there is hydatidiform degeneration of the chorion, or "hydatid mole." This can only be arrived at with certainty first of all by the discovery of cystic villi in the discharges; secondly, by the fact that the uterus is a good deal larger than it ought to be at the time one believes the pregnancy to be advanced; just the opposite to what occurs in apoplectic ovum or fleshy mole, where, though we gather that the woman is three, four, or five months pregnant, we find the uterus is only of the size usual at six weeks, and that, if anything, it is getting smaller. But when the chorionic villi thus degenerate and multiply, we may find the uterus enlarge to the size of a six or seven months' pregnancy by the end of four months.

I think the commonest cause of all, as regards hæmorrhages directly due to the pregnancy, is "catarrhal decidual endometritis." It is general in women who have previously had chronic endocervicitis or endometritis, possibly gonorrhœal in origin. Or there may be a syphilitic taint, even of a congenital origin. Women become pregnant, and then begin to lose a little brownish-red fluid, and then cease to believe themselves to be pregnant. This is the class of case wherein more mistakes are made than in any other. The patient's statement that she is not pregnant is accepted, some superficial examination is made, nothing in the form of abdominal tumour is felt because no bimanual examination is made, and the case is treated with a few doses of ergot; with the result that the uterus, already holding its contents very feebly, expels the ovum. Diagnosis must be arrived at mainly by bimanual examination; the uterus is found to be of the size one would expect, and soft. The only difficulty is to be sure one can feel the upper limit of the uterus. In almost all these cases where there has not been a previous chronic parenchymatous metritis, the uterus is soft and flabby, so that there is much difficulty in appreciating the feeble uterine contractions. If one can make out, bimanually, the size, "anterior vaginal roof stretching," "compressibility of the lower segment," and general smoothness and softness of the uterus, one can be fairly certain that the enlargement is neither due to subinvolution nor to fibroids.

The only other cases in this group which I need mention are *accidental, unavoidable*, and primary and secondary *post-partum* hæmorrhage; and to complete the list we should have to include the *Lochia*.

Our second group consists of hæmorrhages due to *conditions concurrent with pregnancy*—and pretty common they are. First, there are hæmorrhages due to a *constitutional cause*, such as menstruation. Occasionally women menstruate for the first three periods of pregnancy, sometimes more; and of course that would be hæmorrhage during pregnancy of a more or less constitutional type. Secondly, there are various *local causes* of hæmorrhage during pregnancy. There is *Granular erosion* pure and simple, with the hæmorrhage very slight or only present after coitus or some other accidental circumstance. Then there is that peculiar form of *adenoma* which is fairly common during pregnancy. The whole cervix becomes extremely spongy, with pretty free hæmorrhage the moment it is touched, or even without touching, and the cervix looks like a blood-red sea-anemone with its tentacles lying passive, requiring to be distinguished by its softness and appearance from epithelioma. It disappears spontaneously soon after pregnancy is over, and can be kept under control under ordinary treatment while pregnancy lasts. Then there is *mucous polypus of the cervix*. A condition such as I show you an illustration of gives rise to a good deal of doubt if the woman be only examined superficially. A woman may, perhaps, not miss even one period, yet becomes pregnant, and goes on losing almost constantly (for such growths do not always prevent pregnancy). The uterus is found to be growing rapidly, and the diagnosis not uncommonly is that it is a rapidly growing fibroid with two or three mucous polypi hanging out of the cervix. I remember a case being sent over all the way from Canada to have the appendages removed, or hysterectomy performed, and it was found to be nothing but pregnancy in a woman 41 years of age who had recently been married. The pregnancy had not even been suspected. Next we get *malignant disease of the cervix*. I do not know any case where pregnancy has occurred with malignant disease of the body of the uterus, but early cases of carcinoma of the cervix has often been observed to be present with pregnancy. A woman came to the out-patient department of

this hospital suffering from metrorrhagia, which she had for some months with no pre-existing amenorrhœa. We found a good deal of epithelioma, already beginning to slough and ulcerate around the external os, involving a good deal of the vaginal cervix. She had a large uterus, but I did not suspect pregnancy. I took her in, and, with Dr. Black's approval, removed the cervix by the supra-vaginal method. Even then I did not know she was pregnant. She went on to full term, however, then disappeared from our observation; but I subsequently heard that the affection recurred, and Cæsarean section was performed elsewhere by Dr. Cullingworth seven months afterwards.

It is evident that with such hæmorrhage going on it is extremely easy to fail to recognize a co-existing pregnancy. Then there are non-malignant ulcers of the uterus and vagina from foreign bodies such as pessaries, or from syphilis, lupus, &c.

We now come to our second large group, *hæmorrhages apart from pregnancy*. These I have classified into *constitutional* and *local*, and I have subdivided the local causes into those which are apparent on vaginal examination, and those the exact nature of which is only apparent after uterine dilatation.

First let us consider the *constitutional* causes of hæmorrhage apart from pregnancy. We ought to be very careful to remember that there are such causes. A great many local causes are complicated by, and may originally be due to such a constitutional cause as alcohol. This is about the commonest constitutional cause of hæmorrhage. It is a frequent cause of a woman losing too much at her monthly periods; or the alcoholism leads to cirrhosis of the liver, followed by uterine and other mucous hæmorrhages, and if there is the slightest tendency to hæmorrhage from any local cause alcoholism greatly increases it. There is also a form of granular endometritis, which one so often sees associated with alcoholism, that I think it must be cause and effect. Apart from alcoholism, the constitutional causes are mainly those connected with irregular menstruation, such as may normally occur at puberty or the menopause, and need not be assumed to be necessarily due to serious trouble. Books also give hæmorrhagic diathesis, but one does not find that women who bleed excessively from a cut, or when a tooth is drawn, lose more at their monthly periods than

other people. That is my experience. Chronic heart, lung, and liver troubles lead to uterine hæmorrhages, to relieve the general and portal congestion. Hyperlactation is another cause, but that is usually associated with subinvolution. Sexual excess ought to be named in this connection; also acute specific fevers, purpura, and in the early stages of scurvy and myxœdema. I have seen several cases of myxœdema on account of the menorrhagia, which seems to be one of the *early* symptoms.

Now as to the *local* causes of hæmorrhage. Suppose pregnancy has to some extent been excluded, or that examination is being made with that view, and the practitioner has asked all questions regarding constitutional causes, he should then propose a vaginal examination. I find that this is where the practitioner seems to be at fault. An examination is often not made early enough. Sometimes this delay is due to the woman's refusal, but at all events the frequent result is that when the examination has at length been made, it is too late to remedy the disease. This particularly refers to carcinoma of the uterus, and more especially to cancer of the body where dilatation of its neck is further required to perfect the diagnosis. Therefore in all cases of recurrent uterine hæmorrhages in women over 25, or even cases of watery discharge, the patient should be examined.

Now there are certain causes which are discoverable at once by vaginal examination. The mere passing of the finger enables one to diagnose with certainty several conditions, and if this be combined with bimanual examination one will be able to find or exclude three or four conditions to which the hæmorrhage may be due. Thus one may find any of the following:—A mass in the vagina, which will turn out to be either an *inverted uterus* or *fibroid polypus* of the uterus. The use of the sound and a bimanual examination will clear that up. Hæmorrhage may come from ulcerated inverted vagina or cervix with a procident uteri, or the vagina may become ulcerated by the presence of foreign bodies. Then there is that form of vaginitis common after the menopause, called *senile vaginitis* or *ulcerative adhesive vaginitis*, which is evidenced by the vagina being narrowed, and irregularly bound down by submucous adhesions, or by adhesions of the mucous membrane itself, so that the vagina is thrown into

pockets. You can easily break down these adhesions, and bleeding follows rather copiously. Sometimes the discharge is offensive and grumous. Often a woman comes complaining that for some months she has had a watery, offensive discharge, with a good deal of blood, and there is a certain amount of cachexia from the absorption of these products. You fear that you have got epithelioma of the cervix to deal with, but you pass your finger, and feel these bands, and thus discover that, instead of being malignant disease, it is an extremely simple condition.

Cancer of the cervix is, of course, at once diagnosed or excluded by the examining finger, and so are *mucous* or *fibroid polypi* protruding from the os-externum; but it must be remembered that a small mucous polypus is very soft, and its presence may readily fail to be appreciated by the examining finger. A Fergusson's speculum would, however, at once reveal it. The presence of *fibroids* in the walls of the uterus is diagnosed bimanually. You find the uterus is large, hard and irregular, differing from the uniform enlargement of a *subinvolted uterus*, which is another common cause of hæmorrhage, especially if associated with retroversion. *Pelvic inflammation* is another cause of hæmorrhage which does not seem to be often recognized as such. Bimanually, inflammatory exudations are discovered around the uterus, which is found to be fixed or semi-fixed, and the history should be distinctive. A patient gets an acute attack of uterine inflammation, which, spreading along the tubes, soon involves the peritoneum at the back of the broad ligament on one side, and the period may stop for the time being; followed by irregular hæmorrhages, lasting sometimes for weeks, after which there is generally evidence that the disease is wearing itself out. *Pelvic hæmatocèle* is another cause of hæmorrhage which has to be differentiated from phlegmonous exudations. There is also uncomplicated chronic *salpingitis*, associated with typical tubal pains in the neighbourhood of the crest of the ilium, which helps one to decide what is the origin of the hæmorrhage. Vascular caruncle is also given in books, but rarely causes hæmorrhages, and I must mention ruptured hymen, which may bleed furiously.

We have now very few affections left. We have excluded all the constitutional causes, as well as the causes ascertained by examination with the finger

per vaginam or bimanually, and we have now left only such conditions as are represented on these diagrams, viz., those due to intra-uterine causes, viz., intra-uterine fibroid or submucous polypi; submucous fibroids, carcinoma or sarcoma of the body of the uterus, and granular or fungous endometritis.

To arrive at the diagnosis of these intra-uterine conditions, it is absolutely necessary to dilate up the uterus. In all such cases the uterus is somewhat enlarged. In a case of fibroid, one may assume that such is its nature by the nodules which can be felt outside, but not until dilatation is made can we ascertain whether it is a case of polypus or of fungous endometritis complicating the fibroid. Some years ago, I read a paper elsewhere in which I took a good many cases where hæmorrhages were due to intra-uterine causes, where it had been necessary to dilate to get my finger in. In 88 per cent. of those cases the actual cause of the hæmorrhage was a removable one. It would have been useless to have attempted to deal with them by any other means than dilatation and intra-uterine treatment. That is the routine way in which one should go about hæmorrhages in the genital passages, and it is evident that diagnosis is not complete in this last class of cases without dilating up the uterus.

The rapid method, under an anæsthetic, is infinitely preferable to the slow, dangerous dilatation by tents, and the whole thing can be done in under half an hour. If one puts into the vagina near the cervix a glycerine tampon two hours before the operation, the cervix yields perfectly, because the glycerine causes the cervix to secrete, and a secreting cervix is a distensible one. The introduction of even your little finger into the uterus will enable you to make out whether there is polypus, fungous, endometritis, or carcinoma. Of course the index finger must be used if the small one is not long enough.

This diagram will call to your minds how plentiful is the blood supply to the reproductive organs, and why therefore the uterus is an organ which bleeds so readily.

Now a few words as to the treatment of these various conditions. We will first consider the treatment of hæmorrhages during pregnancy. I do not think it is necessary for me to say much about threatened abortions. If "in-

evitable," antiseptic plugging of the cervix and the vagina, and giving ergot internally, is probably the best method for ending early abortions. Three or four hours later, things are generally found so far at an end that you either find the ovum lying loose in the dilated cervix, or you can shell out the whole thing with your finger. Both "missed" and "incomplete abortion" ought to be treated, in the first instance, by ergot and antiseptic vaginal douches. If, however, there be any septic or septicæmic symptoms, the best way is to dilate rapidly and remove the products of conception found in utero. If there be any kind of mole in the uterus, ergot is generally sufficient to empty it, but if not the case must be treated as missed abortion. Endometritis gravis is best treated by iodide of potassium in five-grain doses, without interfering with the satisfactory progress of the pregnancy. If there be a definite history of syphilis, one would give mercury in addition. It is not easy to say why iodide of potassium does answer so well. In some cases its effect may be due to the fact that there is some congenital syphilis, but that does not account for all instances.

The treatment of local concurrent conditions *in pregnancy* has to be on the same lines as for any other condition. Mucous polypus of the cervix would be twisted off, and its base touched with iodized phenol, or if hæmorrhage occur, by the actual or galvano-cautery. These measures would not interfere with the pregnancy. Erosion would be dealt with in the ordinary way; so also with non-malignant ulceration of the genitals. In malignant disease of the cervix, one would have to be guided entirely by how far the disease had spread. If the disease had already involved the connective tissue, rendering radical treatment useless, one would feel inclined to let it go on, and deliver the child at full term by Cæsarean section. If it were an early case of carcinoma of the cervix, with an early pregnancy the treatment would be to scrape away the carcinoma, dressing the cervix with carbolyzed glycerole of pepsine, which would rapidly remove sloughs, and allow healthy granulations to spring up. In a fortnight, when everything is healthy there, induce abortion, and then, after another fortnight to permit of sufficient involution of the uterus, perform supra-vaginal amputation of the cervix, or vaginal hysterectomy.

We now come to discuss the treatment of hæmorrhages apart from pregnancy.

The constitutional treatment is usually sufficiently obvious if a constitutional cause be found; but if we find a constitutional cause it is not wise to at once conclude that there is not also a local cause. It very often happens that there are concurrent causes; one being superadded to, and perhaps the cause of the other. Thus, if one finds a case of alcoholism, it does not follow that there is no granular endometritis present, and nothing can be so useful to the patient as taking her into hospital or into some nursing home, dilating up the uterus, and curetting the endometrium. Under such circumstances she will be under such good influences as to diet and general environment that they may serve to wean her from the alcohol.

There is not very much to be said as to the treatment of local causes of hæmorrhages apart from pregnancy. When the cause is ascertained, it becomes simply a question of removing it. In uterine inversion, the organ has to be replaced by the ordinary methods (Aveling's repositor or manual method).

Senile vaginitis, the ulcerative form, is best treated by breaking down the adhesions, and touching any raw surfaces which are left with nitrate of silver or pure carbolic acid, keeping the walls of the vagina apart by a loose packing of some antiseptic gauze, and finally borax and lead douches should be ordered. In a week or ten days, under this treatment, the vagina is practically healthy again.

Of subinvolution I have not time to speak now. Pelvic inflammation and pelvic hæmatocele have to be treated on the usual lines. Fibroid polypus of the cervix would be removed by the wire *écraseur*, or its pedicle may be divided by scissors, but a mucous polypus of the cervix should be removed by twisting, or by cautery, as hæmorrhage would surely follow if it were snipped off by scissors, for it must be remembered that the cervical fibres do not retract so readily as the muscular fibre of the body of the uterus.

If the cause of the hæmorrhage be intra-uterine, its treatment may immediately follow its discovery.

This intra-uterine polyp may be removed by the wire *écraseur* or by scissors. Fungous or granular endometritis requires curetting, and malignant disease necessitates vaginal hysterectomy unless

contra-indicated by extension of the disease beyond the confines of the uterus,

The drugs useful for hæmorrhages are : *Ergot*—this is the one oftenest given. It acts by contracting the arterioles, apparently by virtue of its action upon the involuntary muscular fibre; and it has a special action upon the uterine muscle, encouraging tonic contraction. It may sometimes act on the intestines, and some people cannot stand it for that reason. This is well seen when the sucking child gets colic whilst the mother is taking ergot. Occasionally also one sees the effect of ergot on the heart by producing pseudo-angina, and infrequency of the heart's beat, especially in people whose hearts are already incompetent. The arterioles all over the body become contracted, and the heart is thus given more work to do. As a rule, however, ergot can be given for any length of time without any trouble, and its action on all forms of hæmorrhages is by the lessened vascularity which it produces in the uterus through the actual uterine contractions. It thus impairs the nutrition of fibroids by this action and by favouring their extrusion. Ergot ought to be given continuously in all cases of hæmorrhage where it is likely to be of service, beginning, as a rule, on the third or fourth day of the period, and should be left off just before the period comes on, especially if dysmenorrhœa be usually present. Sometimes one can continue the ergot in such cases by adding belladonna. The usual doses are $\frac{1}{2}$ dr. of ext. ergot liq. three times a day, or 1 or 2 gr. of ergotin, or $\frac{1}{15}$ to $\frac{1}{10}$ gr. of the very much stronger ergotin. *Hydrastis* is another very valuable drug, and is somewhat similar in its action to combined ergot and belladonna; for besides producing uterine contraction, and checking hæmorrhage in the same way as ergot, it is a sedative. Half-drachm doses of the tincture answer very well. *Cannabis Indica* is also one of the best drugs we have, but I fancy it is not often given. $\frac{1}{2}$, $\frac{1}{3}$, or even $\frac{1}{2}$ gr. of the extract of cannabis Indica is the usual dose, and it often seems to stop hæmorrhage even better than ergot or hydrastis. If there be pain as well as hæmorrhage, as, for instance, in dysmenorrhœa caused by fibroids of the uterus, cannabis Indica is much more useful than ergot. It can also be given in the form of tannate of cannabin gr. ij to x. The tincture is unreliable. Lately, Dr. Wright, of

Netley, has introduced *chloride of calcium*. It was used many years ago in conjunction with bromide of potassium and chloride of ammonium for fibroids. At that time the action of the chloride of calcium was not understood; it was supposed that it deposited lime salts in the tissues of the fibroid, inducing calcareous degeneration. It does not do that. Dr. Wright has found that it is useful in many cases of hæmorrhage, especially those due to hæmorrhagic diathesis. He found he was thus able to check hæmorrhage, by giving 2 to 3 dr. doses of the British Pharmacopœia preparation (*Liquor Calcii Chloridi*) three times a day after meals for two or three days. Dr. Wright finds its action is due to the fact that the blood coagulates much more readily under its influence. I have tried it in one or two cases in which ergot and cannabis Indica have failed, and it certainly does check the passive oozing of blood which sometimes continues for a week or ten days after the more profuse flooding which occurs in many cases of fibroids, and may prove to be a useful adjunct to the means already at our disposal to tide some of these bad cases over the menopause, and so enable oöphorectomy or hysterectomy to be more often avoided.

Whilst speaking of these special drugs, purgatives must not be omitted. They are of the greatest possible importance in the treatment of all uterine hæmorrhages, and should invariably form part of the measures adopted.

REMARKS

ON

THE THEORY OF JAUNDICE.

Made at St. George's Hospital, November 27th, 1895.

By H. D. ROLLESTON, M.A., M.D., F.R.C.P.

Assistant Physician and Lecturer on Pathology in the Medical School.

JAUNDICE is usually divided into obstructive and non-obstructive varieties. Those cases in which there is no manifest obstruction in the larger ducts

are often described as being hæmatogenous, or due to changes not in the liver (hepatogenous) but in the blood. An exception to this general view is the jaundice of biliary, or, as it is often called, hypertrophic cirrhosis. The term, hypertrophic, though clinically useful as differentiating this form from the atrophic cirrhosis described by Lænnec, and exemplified in the bodies of persons devoted to whisky and gin, is somewhat inexact, inasmuch as a cirrhotic liver may be increased in size from changes other than those of biliary cirrhosis. Thus in the mixed livers where fatty change in the hepatic cells is associated with much interstitial fibrosis, the organ is greatly increased, both in size and weight, and it is probable that in the earlier stages of what eventually becomes a typical atrophic or hobnail liver, the size of the organ is increased. But to return, after this digression, to the subject of jaundice. Confusion may result from the use of the term "hæmatogenous" as synonymous with non-obstructive jaundice, if by "hæmatogenous" is meant that the jaundice is solely and entirely due to changes in the blood, and that the liver is quite passive. Jaundice is due to bile in the blood and tissues of the body. In animals in whom life is possible without the liver, it has been shown that bile is not produced after the removal of that organ, and therefore that jaundice cannot be produced in the absence of the liver. It is on the ground that "hæmatogenous" implies the production of bile in the blood independently of the liver that the use of the term is objectionable when applied to those slighter forms of jaundice, which undoubtedly occur in and are secondary to profound blood changes, such as pyæmia, septicæmia, etc.

In these diseases it was thought that bile was produced in the blood, from the breaking-down of the red blood corpuscles, in such quantities that the liver was unable to remove it, and that from the excess jaundice resulted. Now in the lower animals bile is only manufactured in the presence of the liver, and there is no proof that it is otherwise in man. Dr. Hunter has shown that this toxæmic jaundice is in its mechanism essentially obstructive. It is true that the primary factor is the presence of a poison in the blood which destroys the red blood corpuscles, and thus supplies the liver with an excess of hæmoglobin—the antecedent of the bile pigment. An increased output of bilirubin by the

hepatic cells results, but this is followed by a catarrhal inflammation of the smaller bile ducts, and eventually by obstruction. The poison excites the lining of the smaller bile ducts to the production of a viscid mucus which blocks the ducts, and the bile is then, as Vaughan Harley has shown, absorbed by the lymphatics and carried into the general circulation. These experiments further throw some light on the distinction long ago laid down between hæmatogenous and hepatogenous jaundice, viz., that in the former bile pigment only appears in the urine, while in obstructive jaundice both bile pigment and bile acids are present—for the increased production in cases of so-called hæmatogenous jaundice is not of bile as a whole, but of the bile pigment which when absorbed appears in the urine.

In acute yellow atrophy the parenchyma of the liver is rapidly destroyed by an extremely acute inflammatory process, the cause of which is as yet unknown. Post-mortem there is no obstruction in the ducts; they contain some mucus, but no bile. Here it might be thought the jaundice was undoubtedly hæmatogenous in origin, since the disorganized hepatic substance is unable either to manufacture or to excrete bile. This form of jaundice can, however, be explained as being obstructive. The jaundice is an early symptom, precedes the cerebral symptoms, and is due to the spread of inflammation to the ducts, or to inflammation and spasm obstructing the lumen, while the liver still continues to manufacture bile. The bile therefore passes into the lymphatics, and jaundice results from staining of the tissues. As the destructive changes progress in the liver, the formation of bile gradually ceases. The jaundice remains or perhaps diminishes slightly, but death supervenes before it has had time to disappear.

Sudden emotion or nervous shock appears occasionally to give rise to jaundice, and it was at one time thought that this was due to a marked fall of blood pressure in the blood vessels of the liver, which allowed the bile to pass from the duct into the hepatic veins. Vaughan Harley has shown that the bile when re-absorbed passes into the lymphatics and through the thoracic duct into the general circulation, so that this explanation must be given up. It is probable that muscular spasm of the ducts, analogous to the

peristaltic contraction of the intestines set up by fear or violent emotion, occurs, and thus gives rise to a temporary obstruction, as a result of which bile passes into the lymphatics, and so into the general circulation.

In long-standing obstructive jaundice the dilated bile ducts contain only watery mucus; the bile is still formed by the liver, as shown by its presence in the hepatic cells, but being prevented by the heightened tension from entering the duct, passes at once into the lymphatics.

Obstructive jaundice may be due, (1) to some blocking of the lumen of the ducts—a calculus, inspissated bile, a piece of hydatid membrane which has passed out of a cyst, or in very rare cases a round worm which has passed up from the duodenum, or even the liver fluke *distomum hepaticum*, which is so common in the bile ducts of sheep.

(2) To changes taking place in the walls of the ducts. Ordinary catarrhal jaundice spreads up from the duodenum, and by producing swelling of the mucous membrane obstructs the outflow. As has been seen, a catarrhal inflammation may start in the small bile ducts and may spread down towards the duodenum—the bile itself containing the irritating poison. Ulceration due to damage inflicted by the passage of a calculus may result in cicatricial contraction. In a few cases the duct has been found to be congenitally stenosed. In some cases possibly syphilitic fibrosis or gummata may occur in the wall of the ducts. Primary new growth in the bile ducts necessarily obstructs the lumen; when it occurs it is a columnar-celled carcinoma. By continuity of growth primary malignant disease of the gall-bladder may spread along and infiltrate the common bile duct.

(3) Lastly, pressure exerted from without on the ducts. Under this heading the jaundice met with in biliary cirrhosis may be included, the smaller ducts being compressed within the liver. The slight jaundice met with in common cirrhosis may be partly due to the same factor, but is perhaps more often the result of duodenal catarrh. Inflammatory processes, glands enlarged from new growth, or tumours involving the hepatic ducts in the portal fissure, will lead to jaundice, but the gall-bladder will not be enlarged, as will be the case when pressure from without is brought to bear on the common bile duct. A long list might easily

be made of the various abdominal tumours that may thus lead to jaundice. One of the most frequent is malignant disease (scirrhous, spheroidal-celled carcinoma) of the head of the pancreas. This disease necessarily runs a rapid course, since by obstructing *Wirsung's* duct it markedly interferes with digestion, and at the same time, by compressing the bile duct, produces a condition of biliary toxæmia or cholæmia.

REVIEW.

Clinical Lectures on Diseases of the Nervous System. By W. R. GOWERS, M.D., F.R.S. (J. and A. Churchill.)

This is a collection in book form of twenty lectures delivered by Dr. Gowers on various occasions and published in magazines, most of them in the *CLINICAL JOURNAL*. We have read them all with the greatest interest and instruction, each lecture emphasizing some old principle apt to be overlooked, or detailing some new points of interest in diagnosis and prognosis. As some might mistake the title to mean a comprehensive review of the nervous system, we think it best to mention individually the subjects treated of, assuring our readers that each point is dealt with in Dr. Gowers' usual masterly and complete method. Lectures I. and II. are concerned with general points in diagnosis; argyria and syphilis, syphilitic hemiplegia, and bulbar paralysis have each a lecture devoted to them; then follow two lectures on facial paralysis and its sequelæ, one on acute ascending myelitis, and two on locomotor ataxia. Foot clonus and its meaning, syringo-myelia, and the treatment of muscular contraction occupy a lecture each. Lectures XIV. and XV. are on the infantile causes of epilepsy. Lectures on neuralgia, lead palsy, and saturnine tabes come next; and the book concludes with two most interesting and instructive lectures on optic neuritis, based chiefly on a case of gross intercranial trouble causing the lesion. A careful perusal of them is essential to anyone wishing to be up to date in the subjects treated of.

THE CLINICAL JOURNAL.

WEDNESDAY, DECEMBER 25, 1895.

CLINICAL LECTURE

ON

A CASE OF PONS LESION,

BY

ALEX. JAMES, M.D.,

Physician to the Edinburgh Royal Infirmary.

GENTLEMEN,—The case which I am about to bring before you to-day is one which illustrates in itself some very interesting features, and which contains others which may be of value to you in the diagnosis of nerve lesions.

The patient is a man, R——, 57 years of age, who was admitted June 20th, and examined on June 21st and following days.

He complains of giddiness and difficulty in walking and general weakness, of twelve months' standing; of numbness and loss of feeling on the left side of face, arm, leg, and trunk, of four months' duration; of inability to open the mouth widely, three months' duration; and of loss of power on the right side of the face, with double vision during the past seven weeks.

Family History is fairly good. His father died at the age of 55 of some chest complaint. His mother died of "old age" at 88. Three other members of the family died respectively from heart-trouble, inflammation of the bowels, and small-pox. Others are alive and well.

Patient has had seven children, all alive and healthy. He has always had a comfortable home and good food, and is moderate in the use of alcohol.

At his work (a mason) he is often exposed to the weather.

Previous Health.—There is no history of syphilis. He seems to have been perfectly well till thirteen years ago, when after some heavy work and exposure to cold, he states that he came home one night suffering from hoarseness of voice and uneasiness in the throat. Next day he went to work as usual and the hoarseness became worse. At night his voice was gone entirely. He consulted a

doctor and his throat was painted and some local remedy applied, but after about a month, finding himself no better, he came to the Royal Infirmary, Edinburgh, and was admitted to Dr. Brackenridge's ward.

His condition was diagnosed as paralysis of the left vocal cord, and it was thought probable that it was due to a small aneurysm of the aorta involving the left recurrent laryngeal nerve. He was told to be careful in his living, and was sent out a little better. He comes back again to us after thirteen years, and, as we shall see, he still has paralysis of the left vocal cord.

From this time (thirteen years ago) he remained in good health until a year ago, when he began to be troubled with giddiness in walking, causing him to stagger as if drunk.

He noticed that he was obliged to keep his eyes fixed on the ground in front of him to prevent himself from staggering. The tendency to stagger was most marked towards evening; it was not so obvious in the morning.

As a result of this giddiness he once fell while stooping to lift a block of stone at his work.

He gradually became worse, and next he noticed that his general health was becoming somewhat enfeebled.

Shortly after this giddiness began, that is to say about a year ago, he had an attack of vomiting which came on without any known cause. This lasted a week, was of considerable severity, and during it all food was rejected immediately.

Since then he has occasionally had slighter attacks of vomiting. They come on in the morning generally before, or it may be after, breakfast, and are preceded only by a slight feeling of nausea.

About this time also he noticed that he was troubled with headaches which, however, were not of a very severe form.

About four months ago he became aware of, as he describes it, a "numbness, tingling, and coldness, and sense of strangeness," on the left side of the face and body and on the left arm and leg, and a little later than this he noticed that in touching things with those parts he could not feel them so well.

A month after this he found difficulty in opening his mouth widely. His attention was drawn to this by the fact that he was obliged to use a smaller spoon than formerly.

his right eye did not close properly. He says he noticed this, because, when lying in bed with his eyes shut, as he supposed, he still saw light.

He states also that the vomiting and the feeling



Present condition of the patient.

Seven weeks ago he noticed a loss of power in the muscles of the right side of the face, and then he began also to see double, indicating a paralysis of some of the eye muscles. With the paralysis of the muscles of the right side of the face he noticed that

of weakness have been getting worse during the last four weeks.

He was seen about a month ago, prescribed for, and told to return in a month. This he did, and was admitted to ward XXX.

Present Condition.—The patient is a man 5 ft. 6½ in. in height, and at present weighs 10 st. 4 lbs., but he used to be about 12 st. when in health. His muscles are soft and flabby. The right half of his face is absolutely motionless. There is no wrinkling of the forehead, and no reflex closure of the lid on touching the conjunctiva of the right eye. The eye is widely opened, and the conjunctiva is slightly inflamed. The lower lid is also a little removed from the globe, but not much.

There is also evident hollowing in the right temporal fossa, due to paralysis and wasting of the right temporal muscle.

Alimentary System.—The tongue is not in any way paralyzed, but can be moved freely in any direction. He complains of dryness of the mouth, especially on the right side. This is important, because with the portio dura, as you know, you often have the chorda tympani involved, and so salivation affected.

During mastication the food tends to collect on the affected side of the mouth in the hollow of the cheek, and he has to clear it out with his finger. The palate shows some indications as if its right side were a little lowered, but this is not very well marked. There is no difficulty in swallowing.

The bowels are very constipated.

Physical examination of the abdomen reveals nothing worthy of note.

Hæmopoietic System is normal.

Circulatory System.—Pulse 88, strong and full. The vessels are a little thickened, but there are no indications of aortic aneurysm.

Respiratory System.—There is a slight cough and spit. The left vocal cord is quite paralyzed, as already stated.

Integumentary System.—This is normal, except that there is a slight tendency to œdema of the legs.

Urinary System.—Patient has generally to rise once at night to pass water. The act of micturition is at times slightly delayed, not sufficiently to be of importance.

Sp. gr. of urine, 1020; no albumen; no sugar.

Nervous System (Sensory).—Patient complains of coldness, numbness and tingling, as if the parts were "asleep" in the left arm and leg, and on the left side of the body and face. This is specially marked on the arm and front of the chest. There

is some impairment of feeling all over these parts. The area of incomplete anesthesia is limited by the middle line of the body. There is no hyperæsthetic part.

On the right side sensation is perfectly normal, at any rate as regards the trunk and limbs. On the right side of the face there seems to be a little hyperæsthesia, but it is difficult, if not impossible, to determine this, since we have only the defective side to compare it with.

Sensation of locality is impaired on the left side. He cannot say exactly where you have touched him.

There is also some impairment of sensation in the left conjunctiva and in the mucous membrane of the nose and cheek on the left side. Ammonia held to the nostrils stimulates the right more than the left.

Sensibility to weight is not affected.

Temperature sense is impaired on the left side also. Cold is often mistaken for hot, but hot is never mistaken for cold.

Sensibility to electric current on the left side is also diminished, and also sensibility to pain.

Rate of nerve conduction on the left side is diminished.

Muscular sense on the left side is not much affected.

Special Senses.—Smell.—The sniffing movement of the right nostril is not well marked, but he smells such a substance as clove oil equally well on each side. As just stated, ammonia is perceived better on the right side, not so well on the left, since it stimulates the nerves of common sensibility.

Hearing.—He hears constant noises in the right ear, and loud sounds are sometimes painful. But the ticking of a watch is not heard so well with the right ear as it is with the left.

Sight.—The right eye points over to the left, and is completely paralyzed with respect to the external rectus.

The left eye moves well enough to the left, but only a little to the right. Hence there is modified paralysis of the internal rectus of this eye.

Dr. Sym examined the eyes, and the following is his report:—

"There is paralysis of the right movement of both eyes. It is rather a paralysis of movement than of muscles. The internal rectus of the left

side makes no effort to contract when the patient is asked to look at an object moving to the right, but it feebly contracts when he is told to converge for a near object. The other muscles are free. There is homonymous diplopia. Fundus seems healthy. Disc is a little grey. There is no appearance of former neuritis. Field of vision is fairly good. Pupils are of medium size, and contract to light and accommodation. This erroneous projection leads to great giddiness in standing or walking."

Taste.—We have had some considerable difficulty in satisfying ourselves as regards the condition of taste in this case. In the first place, the patient cannot open his mouth very widely, so it is difficult to get at the back part of the tongue. In the second place, we have to contemplate the possibility of the existence of lessened common sensibility on the left half of the tongue, and of this causing some confusion. But all our observations go to indicate that on the whole of the right half of the tongue, front and back, there is a diminution both of taste and of common sensibility. When, for instance, a little quinine, or sugar or salt is applied to the tongue, or taken into the mouth, he distinctly tells us that he feels the taste only on the left side, and all over it. Similarly, when the tongue is touched or pricked with a needle, he tells us that he feels it distinctly better on the left side. This is not altogether what we should have expected, but it certainly is what he says he feels.

Motor.—Organic reflexes are quite normal. Skin reflexes do not in any way deviate from health, but the conjunctival reflex is, of course, absent on the right side.

With regard to the voluntary muscles, there is nothing very noticeable, except with regard to the muscles of mastication. The masseter and temporal are paralyzed and atrophied on the right side, and they, with the facial muscles, show the reaction of degeneration. As regards the pterygoids: we find that the lower jaw is moved slightly over to the right side when it is depressed, and that the patient cannot so well perform a lateral movement of it to the left side. This indicates paralysis of the right external pterygoid muscle.

Co-ordination.—The patient performs movements, such as buttoning his coat, perfectly well.

He can stand with his eyes shut and his heels

together without swaying. When walking with his eyes open he tends to go to the left, and in attempting to correct this he staggers. He has great difficulty in going up and down stairs, on account of the confusion arising from his double vision.

With one or both eyes shut, he walks much better, and can even walk backwards fairly well.

So much for the symptoms and morbid appearances. What we have to do now in connection with the consideration of the case is to make up our minds first as to the locality, and secondly as to the nature of the lesion.

Let us take first of all the paralysis of the face. This paralysis of the face, you can see at once, must be due to a lesion of the nerve itself, or of its nucleus. It is not due to a lesion above the nucleus.

We are inclined to think that it is the *nucleus* of the facial nerve that is affected.

I think with these rough diagrams you will be able to follow me.

The nucleus of the portio dura or facial nerve (F) lies in the lower part of the pons Varolii, and we believe that we have a lesion somewhere at the lower part of the pons involving this root. But a lesion involving the root of the facial must be quite close to the root of the sixth nerve (VI), and it is very likely to involve it as well, and this would explain the paralysis of the right side of the face, and the paralysis of the external rectus of the right eye.

But what about the left eye? This may be explained by the connection of the sixth nerve on one side with the third nerve on the opposite side, so that such a lesion is apt to interfere with the co-ordination of the sixth and third nerves. Dr. Sym's report, as we have seen, indicates this as the explanation.

Further, a lesion here will also be very near the motor nucleus of the fifth (V_m.) nerve which supplies the muscles of mastication. Hence the paralysis of these muscles can also be explained.

As regards the giddiness. Notice that this may be explained as being due to the disturbed working of the eyes. It may be entirely due to this, but it is possible that it may be due in part also to some of the fibres of the portio mollis being involved. Thus we know that from a little lower down than the positions of the facial, sixth, and motor fifth

nuclei the portio mollis or auditory nerve arises, and we know also that in connection with it there are two nuclei of origin. We know also that whilst one of these nuclei seems to be mainly connected with fibres from the cochlea, and so to be

facial, the sixth, and the motor fifth, is very likely to involve this nucleus also. In this way, therefore, might be explained in part the giddiness. No doubt it and the equilibration disturbance are largely due to the eye conditions, but he tells us

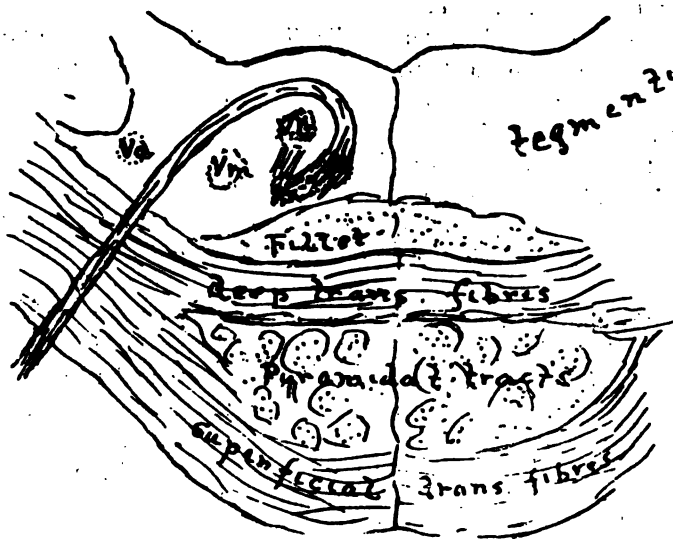


Diagram of section of the lower part of the pons showing the connection of the facial sixth and motor fifth nerves (mastication).

F. Facial nerve. VI. Sixth. Vm. Motor of fifth.

concerned in hearing, the other is mainly connected with the semicircular canals and vestibule, and is supposed to be concerned in equilibration. In the

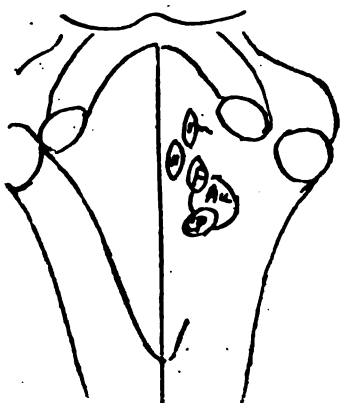


Diagram showing the relative position of the nerve nuclei below the floor of the fourth ventricle.

F. Facial. VI. Sixth nerve. Vm. Motor of fifth.
Au. Portio mollis. Gp. Glosso-pharyngeal.

diagram they are both indicated by the area marked Au, and it is evident that a lesion involving the

that even when he sits quietly with his eyes closed he still feels the giddiness to some extent.

Hearing.—As you are aware, in paralysis of the facial nerve hearing is often affected owing to the action of the stapedius muscle being interfered with. In this case we have also, as just stated, to take into consideration the possibility of the auditory nucleus itself being involved.

Next, as regards the loss of sensation on the left side of the head, trunk, and left arm and leg.

The sensory nerves from the left leg, arm, and side of the trunk pass into the posterior part of the cord, and having decussated either in the cord or in the medulla, pass through the tegmentum of the pons; up the crus to the brain. We can understand, therefore, that such a lesion will affect them and account for the hemi-anæsthesia.

Further, to account for the impairment of sensation on the skin and mucous membranes of the left side of the face, we have to suppose that the lesion on the right side of the pons, while leaving unaffected the sensory part of the fifth nerve on that side, is in one way or other interrupting the

continuity of the fibres which convey impulses from the sensory part of the fifth on the left side, upward to the right side of the brain.

Taste.—As regards taste we have seen that in our patient not only is this special sense impaired over the right half of the tongue, but that common sensibility appears to be also impaired in this situation. The loss of taste is explained by the view that the glossopharyngeal is the sole sense of taste, and that it supplies the back of the tongue through its main trunk and the front part through the *pars intermedia* and chorda tympani. Hence in our patient we must suppose that the glossopharyngeal nucleus or fibres of origin are involved.

But how can we explain the impairment of common sensibility on this right half of the tongue with no other evidence of lesion of the sensory fifth nerve on that side? Explanations might be hazarded, but they could only be conjectural.

Larynx.—As regards this the question might be asked, Can the paralysis of the left vocal cord—which was noticed thirteen years ago and which is now very much as it seems to have been then—be a part of his present disease? I believe not. Do I neither think that it is due to aneurysm. Probably it is due to some inflammation of a rheumatic nature, which has led to adhesions between the left arytenoid and parts around.

As regards locality of the lesion in this patient, therefore, I believe it to be situated in the right side of the lower part of the pons, involving the nuclei of origin of the facial, sixth and motor fifth nerves, extending downwards so as to implicate to some extent the nuclei of the glossopharyngeal and portio mollis, and upwards so as to implicate the sensory fibres which have crossed from the left side of the head, trunk, and limbs.

Nature of the Lesion.—This is obscure, but since the condition has been going on steadily for twelve months, I think that probably it is of the nature of an infiltrating glioma. It is of slow growth, and, although there is some headache and vomiting, there is no distinct optic neuritis. Still, I think it is of this character.

Treatment.—It is not necessary to say much about this. We are giving him the iodide and looking after his general health. This is all we can do at present.

A LECTURE

ON

NEW GROWTHS OF THE PENIS.

Delivered at St. Thomas' Hospital, May 31st, 1895.

By Sir WILLIAM MAC CORMAC, M.A., D.Sc.,
F.R.C.S.,

Emeritus Lecturer on Clinical Surgery at the Hospital.

GENTLEMEN,—The subject of my remarks to-day is that of new growths affecting the penis. I will first show you two cases from the wards.

The first I have to present is a man 59 years of age; the penis was amputated three and a half years ago for epithelioma. He remained well for a considerable time, but a warty growth is now evident in the cicatrix, with a certain amount of glandular swelling in both groins. The operation appears to have been through the pendulous portion of the penis. The second case is that of a man 75 years old. Some four years ago he first noticed a discharge under the foreskin which had an offensive smell, and about twelve months ago the glans got very painful; he also noticed a lump, for which he procured a lotion from a dispensary. He has lost a good deal of flesh, and there appear to be a considerable number of enlarged glands in each groin. At the junction of the prepuce and the glans there is a large superficial ulcerated swelling, and there is phimosis.

In connection with the subject, I would like to remind you of a few points of anatomical interest. The penis is a complex organ, composed of a corpus spongiosum on the under surface, and two corpora cavernosa above. The corpora cavernosa are cylindrical bodies placed side by side; they are intimately connected for the anterior three-fourths, but separated behind, and they form two tapering crura connected to the rami of the pubes and ischium. These two bodies form a rounded extremity, in front, which is received in a sort of cup-shaped hollow at the base of the glans penis; posteriorly they diverge, and at their origins are so closely adherent to the rami of the pubes and ischium, that it is difficult to separate them from the bone for purposes of operation. Each cavernous body has a special envelope, and the three constituent parts of the penis have a

common one. That surrounding each cavernous body consists of strong internal circular fibres, while the common envelope is composed of white and yellow elastic tissue. The septum between is thick and complete posteriorly, but incomplete in front; fibrous bands proceed in every direction from the internal surface of the fibrous envelope, forming separate compartments. These trabeculae present on section a spongy appearance, and consist of white fibrous, yellow elastic, and muscle tissue, which contain numerous vessels and nerves. The spaces are largest in the centre, filled by venous blood, and lined by an endothelium resembling that in the veins. The fibrous partitions are larger and stronger at their periphery than elsewhere, and the cavernous spaces communicate freely. Numerous channels carry the lymphatic fluid from the penis to the lymphatic glands. There are two sets, superficial and deep. The superficial drain the glans, prepuce, and anterior portion of the mucous membrane of the urethra; a median dorsal trunk receives vessels from both sides of the penis; and in addition to these, four or five lateral vessels run back on each side. Near the pubes the median trunk bifurcates, and, joined by the lateral vessels, runs to the innermost group of inguinal glands, and also to the glands on the inner side of the saphenous opening. The deep lymphatics drain the corpora cavernosa and the corpus spongiosum, and pass beneath the pubic arch, to enter the deep lymphatic glands of the pelvis and those along the external iliac artery.

The arteries and veins derive interest from the fact that four or five of them frequently have to be tied or controlled during operative procedures. There are the two dorsal arteries of the penis from the internal pudic, and the arteries of the cavernosa. The blood from the skin forms a superficial dorsal vein, which is subcutaneous. The blood from the cavernous spaces passes into veins which converge on the dorsum to form the deep dorsal vein, the latter lying in the median furrow between the corpora cavernosa immediately beneath the fascia. Great numbers pass at the root of the penis to join the prostatic plexus and the plexus of Santorini. The nerves supplying the penis are derived from the internal pudic nerve and the hypogastric plexus.

New growths may be either benign or malignant. Of benign growths of the penis, the follow-

ing have been met with: cystic, fibrous, horny, sebaceous, and fatty. Besides these, naevoid growths are encountered, both of a cavernous and capillary character. The latter are sometimes seen on the glans penis, and the cavernous kind perhaps more frequently in the body of the organ. Bony formations in the septum (the normal condition in some animals) occasionally occur; and calcareous plates may develop in the same situation or in the fibrous envelope.

Two kinds of cysts are met with, sebaceous and dermoid. The former may be a retention cyst of a sebaceous gland, or it may originate in an accumulation of secretion in a cyst-like cavity formed by the adhesions which are so common between an unretracted prepuce and the glans penis. In the operation of circumcision one often notices how closely adherent the prepuce is to the glans, sometimes to such an extent that it may be difficult to separate them. Dermoid cysts are very rare; the majority of them have been found along some part of the raphe, on the interior surface of the organ, where the two halves naturally unite. Fibrous tumours occasionally develop in the septum or in the sheath of the corpora cavernosa, but they are rare. It is a curious fact that gummata are very rarely met with in the penis.

By far the most common kind of new growth in the organ is a papilloma, frequently attributable to uncleanness, and often found in cases of phimosis. Retained secretions help to cause these growths, and they appear in cases of gonorrhoea. The warts may be scattered over the surface of the glans, or surround the corona and form large masses which sometimes produce appearances closely simulating those of malignant disease, cause perforation of the prepuce, and sprout through it in one or more places. The difficulty in discriminating between these and malignant growths is often great. Under ordinary conditions a correct diagnosis may be made by remembering that a simple papilloma or wart does not invade the structure on which it lies, hence the tissue immediately adjacent will be normal in character and consistence; there will be no hardening or anything to suggest infiltration, and it does not tend to ulcerate. A very important factor in the diagnostic discrimination of these growths is that when the disease is warty other smaller papillomatous elevations will be seen on

other parts of the glans and prepuce, a condition not found in malignant growths.

The treatment of these warty tumours is a simple matter. They may be clipped away with scissors, and it is desirable to apply perchloride of iron solution, or strong carbolic or nitric acid, to the base of the growth to make sure it is thoroughly destroyed. Small warts may have one of these caustics or strong acetic acid several times applied, when they will shrivel and drop off. Cleanliness must be subsequently observed, as neglect of this will surely lead to recurrence of the trouble. An effort should be made to secure exposure of the glans penis by permanent retraction of the foreskin or, if necessary, by circumcision.

Malignant growths of two kinds are met with in the penis; one is common and the other rare. I need scarcely tell you that the common form of malignant disease is epithelioma or squamous-celled carcinoma. Sarcoma, the other form, is very rare indeed. I have here, from the museum, eight or nine specimens of epithelioma of the organ. They are all of the squamous-celled variety. The disease generally appears near the corona, either upon some part of the glans penis or the inside of the prepuce, near the corona. It may also invade the body of the organ, by extension from the urethra or glans.

The age at which the disease occurs is somewhat later than in most forms of cancer elsewhere. It is rare before 40, and most frequently appears between the ages of fifty and sixty. Kauffmann states that one-third of all cases occur at this period of life, while Demarquay and Kauffmann found that 75 per cent. of the cases were between 40 and 70 years old. Ricord says that most cases occur from 60 to 70, and he never saw one younger than 40. The nett result of various statistics is that of all cases of cancer, about 3 per cent. occur in the penis.

It may arise in two different ways. Most commonly it commences as a hard, warty growth, whose surface soon ulcerates, and the base speedily becomes indurated. This warty growth is fixed, and quite different from a soft superficial papilloma. It furnishes from the ulcerated surface a peculiarly foul-smelling secretion. It may also commence as a small lump, which may be an infiltration of a lymphatic plexus. This gradually becomes larger and ulcerates on its raised surface, and does not

materially differ, when the disease is somewhat advanced, from the other form.

Cases of what is called scirrhus of the corpora cavernosa penis are described in books. Primary carcinoma cannot occur in this situation except by extension from the glans or urethra. It is possibly an alveolar carcinoma originating in the mucous follicles of the urethra, or might as a rare occurrence take place in Cowper's gland.

Epithelioma may be the result of a cancerous transformation of simple warts; that is, the columns of epithelium penetrate the basement membrane and invade the deeper structures. A very considerable proportion of cases of cancer of the penis are met with in subjects of habitual phimosis, who at the same time do not, or cannot, keep the parts clean, the decomposing secretions proving a source of continual irritation. It is calculated that as many as 65 per cent. of those affected have a phimosed prepuce. In the preparations before you, in the large number of specimens of epithelioma penis preserved in the Hunterian Museum, the great majority show evidence of a condition of antecedent phimosis. Mr. Travers, a surgeon to this hospital, was one of the first to point out that Jews are hardly ever the subjects of cancer in this situation. This, of course, is not a conclusive argument, as the number of Jews in this country affected by cancer of the penis would not be large, Jewish patients not being numerous, but you may take the statement for what it is worth. The French surgeon, Demarquay, relates sixty-two cases of penile cancer, of which forty-two were the subjects of phimosis. Another authority, König, goes so far as to say that he considers phimosis to be the most prolific cause of cancer.

Whilst the disease is generally met with in people comparatively old, it does occasionally occur in younger ones, and in such it appears to be largely attributable to the fact that the foreskin is non-retractile. When a chronic foul-smelling discharge is found exuding from beneath a phimosed foreskin it should lead to a suspicion that the malady may be malignant, and the foreskin should be slit up and the parts underneath carefully examined. There seems to be some difference of opinion as to the predisposing influence of the syphilitic poison on the production of this disease. There cannot be much doubt that the presence of scar tissue on the

glans penis will, by reason, of its being less capable of resisting injurious influences to the same extent as normal skin, predispose to the formation of cancer. A German surgeon, Schuchardt, has described a condition in the glans penis which he calls preputial psoriasis, as an antecedent or pre-cancerous state, that is, one tending to favour epithelial in growth. The inception of the disease has been attributed to repeated connection with a woman suffering from cancer of the os uteri; and recent observations seem to make this view a reasonable one. In a few cases injury has proved an exciting cause of new growth, but this influence is more evident in cases of sarcoma than in carcinoma. The causative influence of phimosis is not accepted by all surgeons; in the latest edition of Heath's Dictionary, Swain does not favour this view.

There is not much to be said about the progress of the disease. When ulceration begins, the course is similar to that of epithelioma elsewhere; the ulcer becomes excavated, the base hard and covered with a yellow foul-smelling secretion, and the loss of tissue is considerable, resulting in the gradual destruction of the glans. Curiously enough, the urethra remains for a long time intact, so that the subject is able to make water with comparative freedom and with little pain, which would otherwise be felt if urine were frequently trickling over the diseased surface. It is also important to note also that for a long time the corpora cavernosa remain intact, and, when the disease does invade them it is by way of extension. This is important from an operative point of view. Before the introduction of anæsthetics, operation was frequently delayed or objected to, the disease continued, and the penis gradually ulcerated off. The inguinal glands are constantly, and at an early period, involved in this disease, and what is extremely important and has not been sufficiently emphasized, is that cancerous foci may be met with in these glands before any external enlargement is evident. Not only do the inguinal glands become involved, but also those along the iliac arteries, and the pelvic glands. The glands near the inner side of the saphenous opening and the long saphena vein are very commonly implicated; they soon adhere to the vein wall, which becomes involved and will require excision, a double ligature being applied to the vein. Metastasis in distant parts is very

rare, in which respect it resembles other epitheliomata. Out of forty-eight cases reported by Kauffmann, the inguinal glands were involved in forty, generally on both sides; of forty cases reported, thirty were on both sides. One side, however, is generally affected more extensively than the other. In twenty-seven out of thirty cases, perhaps, the disease will recur in the inguinal glands in the manner noticed in the man I have just shown you. The local recurrence of the tumour in the cicatrix occurs in about 6 per cent. of the cases.

When the glands are not involved the prognosis is favourable after amputation, but it is very unfavourable when they are affected. The disease is recorded to have been definitively cured in many instances. The pelvic glands are inaccessible unless, indeed, as proposed by König, the falciform and Poupart's ligaments be divided, and the pelvic glands reached in this way. But even where any lasting cure is out of the question, a free removal of the ulcerated growth and a transference of the urethra to the perineum may do much to mitigate the sufferings of the patient.

Sarcoma, as I have said, is very rare. It generally affects the body of the penis, and is of the round-celled variety. It commences either in the septum or in the investing sheath of the corpora cavernosa. Cases of spindle-celled and melanotic sarcomata have been reported by Holmes and by Fischer. The course of the disease is rapid, and it usually involves the lymphatic glands. Metastases are frequent and early, and the prognosis is bad.

There have been recently only two cases; one (a patient in one of my wards) was a case of sarcoma of the corpus cavernosum.

Male, æt. 64.

Growth first noticed six months ago as a constricting band around the penis.

On admission. There is a large, hard tumour the size of the fist situated in the upper part of the right side of the scrotum. Upper limit easily felt, while the lower reached into the scrotum and down to the testis on that side, and stretched far back into the perineum in the mid-line. The root of the penis is very hard and fixed, and the urethra imbedded in the growth which extends to the left of the mid-line. The skin over the tumour is adherent and œdematous; no enlarged glands were felt.

Operation by Sir William MacCormac.

The whole of the external genitalia were removed, and the urethra brought out and fastened in the perineum.

A flap taken from the left side of the scrotum was used to cover the raw surface.

The patient sank and died on the sixth day.

Microscopically the growth was a small round-celled sarcoma, commencing in the right corpus cavernosum, very dense, and with much fibrous tissue in places.

Post-mortem. No glands or secondary growths were found.

In Mr. Battle's case, published in the "Lancet," 1893, a tumour was met with 4 inches by 2 inches, growing from the under surface of the penis, and connected more especially with the left corpus cavernosum. It projected in the perineum and was encapsuled. There were no enlarged glands. The tumour was excised, and the anterior part of the penis not removed. It proved to be a large mixed-celled sarcoma, undergoing myxomatous changes. The man made a good recovery from the operation with a urinary fistula, but he died forty-three days afterwards.

Cross, Fenwick, Tay, Podrazki, and Kobler have also described cases.

With regard to treatment of either form of new growth, there is nothing short of complete removal of the diseased parts. When the disease is limited to the glans, as it often is, the operation may be safely done through the pendulous portion of the penis, always provided you can make the amputation sufficiently in front of the scrotum to prevent dribbling of the water. Where this cannot be done, Thiersch's operation is a very valuable one. It was brought forward in 1875, and consists in transposing the urethra to a button-hole opening in the perineum after the other portions of the penis are amputated. The patient is then able to micturate with great comfort, and there is no subsequent tendency to stricture. He may need to make water like a woman, but even this is not necessary if a small tube be used to collect the water as it flows out. The method introduced by Thiersch enables one to remove the organ further back, and to separate the diseased parts still more completely; the scrotum may be split open, the suspensory ligament divided, and the crura detached. A sufficient length of spongy urethra can

usually be retained, as it is generally infiltrated less extensively than the cavernous bodies, sufficiently long to bring it to an aperture in the perineum, and the scrotal wound closed by suture. Mr. Gould reported a case of this kind in the "Lancet" of May 20th, 1882. He insists on the importance of a complete removal of the crura from their attachments to the ischium and pubes, and the operation has been named the Thiersch-Gould method. But Delpéch, in 1832, appears to have anticipated Thiersch's suggestion, and was the first to do it. It was next performed by L'Allemand in 1844, followed by Bouisson in 1855. In later years it has been carried out by many other surgeons. In Kocher's "Klinik" there were three such cases in 1879, so that the new operation is an old one rediscovered, as has often been the case in the history of surgery.

In an early stage, when the disease is limited to the extremity of the organ, and the penis can be amputated in front of the scrotum sufficiently wide of the disease, the operation is a simple one. The skin should be slightly retracted, made equally tense, and divided by a circular sweep of the knife all round, so as to form a velum of skin. The corpora cavernosa are then cut through at right angles to the axis of the organ; the spongy portion must be separated by a touch or two of the knife for half an inch further forward, and then divided. The edges of the urethra can now be attached to the skin, and, if necessary, the urethra can be divided along its upper aspect for a short distance to allow the suturing to be more complete, and to avoid a subsequent tendency to stricture of the opening.

A method of amputation favoured by some is to dissect back a dorsal square flap of skin, then transfix, passing the knife between the spongy and cavernous bodies, divide the cavernosa vertically upwards, and cut the spongy urethra long enough to bring it through an opening in the dorsal flap, to which it is subsequently sutured, or a lateral flap method may be employed.

If a more extensive removal or so-called extirpation of the penis be desirable, the scrotum may be split open, and the cavernosa with the crura removed close to their attachments to the os pubis and ischia, and the urethra preserved sufficiently long to attach it to an opening in the skin at the middle point of the perineum. The

patient will have the trifling inconvenience of having to make water in a sitting posture, and not this even if he uses a tube. The scalding and distress he would otherwise endure are quite obviated.

One other point I would just allude to is as to the desirability or otherwise of performing castration in cases of complete removal of the penis. This has been strongly advocated by some, as it appeared to them the removal of both testicles was warranted to remove sexual desire. I do not consider this is a justifiable addition to the severity of the operation, and one reason urged in favour of the removal of these organs, that a secondary growth in them is thereby prevented, is scarcely valid, as the occurrence is not at all likely to happen.

A CLINICAL LECTURE

ON A CASE OF

APOPLEXY.

By J. MICHELL CLARKE, M.A., M.D. Cantab.,
M.R.C.P. Lond.,

Physician and Pathologist to the General Hospital; Lecturer
on Practical Physiology at University College, Bristol.

THE case of the man admitted in a state of profound coma two days ago presented several instructive features.

He was a man æt. 53; and the history brought with him was that he was a plumber, and had been engaged that morning in repairing a bath. The bath was empty, and he was found lying in it in a state of complete unconsciousness. Half an hour previously he had been seen, and was apparently in his usual health. When I saw him about two hours had elapsed from the onset of the attack.

Further inquiry of his wife elicited that he had not eaten his breakfast that morning, on account of loss of appetite; but as he often suffered from morning sickness, this attracted no particular remark, and he went off to work much as usual. Seven years ago he had had a fall on the head, with slight concussion, but he had since then suffered no ill effects from the fall, and had never had fits of any kind. He had been a heavy beer drinker, but not for the last two years. He had suffered from several attacks of gout during the last five

years, and latterly had been obliged to get up to pass water two or three times every night. For a short time previously he had complained of a little pain in the heart, with shortness of breath, and slight swelling of the feet towards evening. He had never had syphilis. Although a plumber, the particular work in which he was engaged did not involve much handling of lead.

On examination, the patient was a rather stout man, with a pale, somewhat puffy face. He lay in a state of profound coma, with stertorous breathing. The surface of the body was cold, and there was a slight dusky tint about the face.

Loud rhonchi were heard all over the chest in front, and coarse crepitations at the bases of the lungs behind, otherwise they were normal. The apex beat could not be felt, the heart-sounds were weak but normal, and there was no cardiac murmur.

The abdomen was not distended; there was no ascites; the liver came nearly two inches below the ribs, its edge was smooth and sharp. The bladder was distended, reaching half-way to the umbilicus, but attempts to withdraw urine by catheter failed owing to the presence of a very tight stricture through which the catheter could not be passed. The respiration was 28 to the minute, the pulse-rate 108; the pulse was regular but rather weak, with a small wave, the arteries thickened. Temperature in rectum 95.6°, in R. axilla 96.2°. The coma was profound, no sensibility being manifested during the attempts to pass a catheter.

The head was turned to the right, and if moved towards the left slowly turned back to the right again. The eyes were also directed to the right for the most part of the time, but showed from time to time consensual lateral oscillations of slight extent, during which it was noticed that they never turned to the left beyond the mid-line. Sometimes, however, whilst the right eye was turned to the right the left was directed straight forwards. The pupils were of moderate size, reacted very feebly and slowly to light. Conjunctivæ insensitive. The right eyelid was closed, the left partly open, and the wrinkles on the left side of the face smoother than on the right. The mouth was probably drawn slightly to the right. A faint blue line was detected at the margin of the gums.

There was slight general rigidity of all four limbs, more marked on the left side, especially in the left leg. The superficial reflexes were all abolished. The left knee-jerk was exaggerated, the right normal; there was no ankle-clonus.

No deformities or muscular atrophies were present to indicate the existence of chronic nervous disease.

We had to decide (1) the cause of the coma, (2) if due to an encephalic lesion, the nature and position of this lesion.

The first thing to eliminate in such a case is the presence of injury to the head. The man might have slipped and struck his head against the side of the bath. On careful examination, however, no sign of any bruising or other injury was detected.

The next general cause of coma of primary origin, narcotic poisoning, was easily eliminated; the coma was too profound for alcoholic poisoning, and we also knew that he had taken no drink that day; and the state of the pupils precluded opium poisoning.

The signs of a unilateral lesion—turning of head and eyes to the right, predominant affection of left side of face, and rigidity of left limbs—were sufficient to make it evident that the lesion was encephalic. Had we, then, to deal with hæmorrhage, embolism, or thrombosis? or, remembering the existence of a faint blue line on the gums, might the symptoms belong to that group of symptoms occurring in lead poisoning termed “lead encephalopathy”? In deciding this question the depression of temperature to 95.6° in the rectum, 96.2° in the axilla, pointed to hæmorrhage. The initial depression of the temperature is far more marked in encephalic hæmorrhage than in either thrombosis or embolism. Practically, apart from hæmorrhage, a lowering of the temperature below 96° is only observed in thrombosis of the basilar artery (Bastian, *Paralyses—Cerebral, Bulbar, and Spinal*, p. 71). Embolism could be excluded in the absence of any sign of valvular disease of the heart. In lead encephalopathy, epileptiform convulsions with delirium generally precede the onset of coma, which is rarely complete, and there may be a moderate elevation of temperature. One of the results of chronic lead poisoning is kidney disease, and another gout. The history of gout, of frequency of micturition at

night, the pasty, pallid aspect of the face, the thickened arteries, and presence of the blue line, made the existence of kidney disease, even in the absence of an examination of the urine—which, unfortunately, could not be obtained—practically certain. This being so, was the coma of uræmic origin? The mode of onset and the symptoms precluded uræmic coma. To sum up, such profound coma of sudden onset, with great lowering of the temperature, in a patient over fifty, the subject of kidney disease, would presumably be due to hæmorrhage.

Having gone so far in the diagnosis, the next question to be considered was that of the position of the lesion. With regard to this the unilateral symptoms indicated the right cerebral hemisphere as the seat of hæmorrhage. Absence of convulsions showed that the cortex was not involved. The characters of the coma as above described, and the presence of slight general tonic spasm in the limbs, made hæmorrhage into the ventricle most probable. When this occurs it is generally due to rupture either of one of the anterior branches of the cerebral artery running to the middle caudate nucleus, or posteriorly of one of the branches of the middle or posterior cerebral which pass to the optic thalamus.

The man died about four hours after the onset of the attack. The following were the chief pathological changes found at the post-mortem. On removing the heart the left ventricle was seen to be very large; its walls measured, nearly $1\frac{1}{2}$ inches in thickness; this hypertrophy was much in excess of the small amount of dilatation present. The heart weighed 1 lb. 3 oz. The valves were healthy and competent. The heart-muscle appeared normal. There was thus the characteristic “renal heart.” The liver and spleen were healthy. The kidneys showed marked changes. The right weighed 5 oz., the left 4 oz. The capsule in both stripped badly, tearing the cortex and leaving a finely granular, pale, or mottled surface with some dilated stellate veins. In addition, in the left there were a number of small cysts filled with a dark gelatinous fluid; these cysts were also scattered over the section of this kidney. In both on section the surface was mottled, and the cortex very irregular in extent, in some places being reduced to a proportion of one to four, or less, of the medulla. The cortex was pale, some of the vas-

recta injected, the pyramids were reddish in colour. Both kidneys were extremely tough, not breaking down on firm pressure. The bladder was distended and sacculated; a stricture was found in the membranous portion of the urethra, reducing the lumen to very narrow dimensions. The ureters were not dilated.

On removing the skull-cap there was no sign of fracture or injury to the skull. The convolutions of the brain were flattened, both hemispheres distended, the right especially so, and there was an absence of cerebro-spinal fluid; a little blood had escaped under the arachnoid on to the under surface of the cerebellum and sides of the medulla oblongata. On making transverse vertical sections through the brain from before backwards, the lateral ventricles, third ventricle, iter, and fourth ventricle were found full of blood, the largest amount and the greatest distension occurring in the right lateral ventricle. The blood came from a large hæmorrhage into the right hemisphere, which had occurred externally to the lenticular nucleus. The white matter of the internal capsule, the lenticular and caudate nuclei and optic thalamus were so completely torn up and destroyed by the extensive hæmorrhage as to render it impossible to trace its exact source. The arteries at the base of the brain showed a moderate degree of atheroma. Owing to the feeble action of the heart the apex beat could not be felt, otherwise no doubt it would have been found of heaving character and displaced outwards, and have given additional aid in diagnosis. The case thus well illustrates the events in a large cerebral hæmorrhage bursting into the ventricles in the course of chronic renal disease and the principles on which the diagnosis is arrived at.

A further feature of interest at the post-mortem examination was the discovery at the apex of the left lung of thickening of the pleura, and about one inch below the surface near the posterior border of the lung of a partly caseous, partly cretaceous nodule the size of a pea surrounded by a capsule of fibrous tissue. One other smaller caseous nodule was found near it, and two or three small calcified bodies. Two or three small fibrous bands passed from them in the direction of the thickened pleura, and there was slight general increase of the connective tissue around them. These were undoubtedly the healed remnants of

long antecedent tubercular deposits; we could obtain no history from his wife of cough or of any lung-symptoms. These evidences of healed tubercular lesions are found post-mortem with some frequency. Dr. J. K. Fowler found them in 9 per cent. of 1943 post-mortem examinations, and in the majority of the cases in both lungs. Dr. Leith in 1400 examinations found them in 12 per cent; Dr. Sidney Martin in 12 per cent. in 443 cases; whilst Heitler, of Vienna, gives a lower percentage—4 per cent. out of 16,562 examinations.

A LECTURE

ON

THE EXTERNAL EXAMINATION OF THE EYE.

Delivered at the Royal London Ophthalmic Hospital, in connection with the London Post Graduate Course.

By R. MARCUS GUNN, M.A., F.R.C.S.,

Surgeon to the Hospital; Assistant Ophthalmic Surgeon to University College Hospital.

GENTLEMEN,—I shall divide my lecture to-day into two parts. The first portion I will devote to a consideration of the normal appearance of the external parts of the eye and of its surroundings, and to a short explanation of how to examine them. In the second part we will examine together a number of different cases of ocular disease, with the view of applying these methods of examination to practice.

By the term "external examination of the eye" we here understand any examination that can be made without the aid of the ophthalmoscope, and without subjective testing of vision.

Let us take this lad, as having eyes which are practically normal. I will indicate in him the special features of the orbital margin and eyelids, and then pass on to consider the eyeball as far as we can examine it externally, taking consecutively the conjunctiva, the sclerotic, the cornea, the iris, and the pupil.

The *orbital margin* can be followed all round with the finger, and the characters of its edge

noted. In cases of disease, the corresponding parts of the margin on each side should be carefully compared as to relative sharpness, hardness, prominence, and tenderness. Note that the inner margin is normally rather difficult to follow accurately; this difficulty is due to the presence of a band of fibrous tissue, the *tendo oculi*. If I place one finger firmly over the outer orbital margin, and draw the skin and eyelids forcibly outwards, the *tendo oculi* stands out distinctly, so that its position can be accurately determined. Just below its lower edge a sharp crest of bone is felt, which affords a valuable guide to the position of the lacrymal sac.

The Eyelids.—The upper lid extends from its free edge upwards to the eyebrow. Running across it horizontally we note a furrow, called the superior palpebral groove; this divides the upper lid into two portions, a lower or *tarsal*, and an upper or *orbital* portion. If you take hold of the lid, you can appreciate by touch the line of the upper border of the tarsus corresponding to this groove, and further you can distinguish the presence of the tarsus in the lower portion by pinching up horizontally the whole thickness of the lid between the finger and thumb.

The lower lid has no distinct boundary below, but may be said to extend from its free edge downwards to immediately over the lower orbital margin. In the aged a groove can often be seen running horizontally across this lid also, marking the lower edge of its tarsus, but in this lid it is not discernible; by palpation, however, we can recognize the presence of the tarsus as we did in the upper lid.

On looking at the free margins of the eyelids you can see that the lashes are not arranged in a single row, but in two or three rows running closely parallel with one another. Hence the abnormal condition known as *distichiasis* does not mean strictly that there are two rows, but that there are two series of rows. On slightly everting the lid-margin, you observe that it is fairly thick, and that it has two edges, of which the outer is rounded, while the inner edge is sharp. Along the outer edge the lashes are arranged, and the sharp inner edge corresponds to the junction of the skin and mucous membrane. The space between the two edges is covered by skin, which you see is well lubricated by the secretion of the Meibomian glands, the openings of whose ducts you can observe along this part of the lid-margin.

We now evert the eyelids, so as to be able to

examine their inner surfaces. The lower lid is easily everted by placing the tip of the finger on the skin of the lid and pressing it backwards over the lower orbital margin; the patient should at the same time be directed to look upwards. To evert the upper eyelid, proceed as follows:—Rest one hand, with its thumb pointing downwards, on the patient's forehead, the tip of the thumb being placed in the superior palpebral groove; now tell the patient to look down; take hold of the lashes between the finger and thumb of the other hand, pull the lid gently downwards, and then you will be able readily to evert it over the tip of the thumb lying in the groove. In ordinary cases this is a simple enough manœuvre, and is more easily demonstrated and taught practically than described in words. Having everted the lids, we now see a glistening mucous surface, the palpebral conjunctiva, and through it we can distinguish a number of yellowish lines running from the free edge of the lid towards the upper limit of the tarsus; these are the Meibomian glands, the openings of whose ducts we formerly observed on the lid-margins. These glands lie in the substance of the tarsus, and are liable to become inflamed. You will observe that the outer and inner ends of the conjunctival surface of each eyelid are slightly rougher and more vascular than the rest; this is a normal condition, due to the presence of certain glands. We must become familiar with the appearance presented by this surface of the normal eyelid, in order to recognize diseased conditions. Note also that the conjunctiva is smooth and tightly adherent over the tarsus, while beyond it is more uneven and freely movable.

By placing the thumb firmly on the skin just above the outer canthus, and drawing the lid upwards and outwards and then directly upwards, while the eye is directed downwards, we render visible the lower lobe of the lacrymal gland. The possibility of thus viewing this part of the gland should be remembered.

The *palpebral aperture*, or opening between the upper and lower lids, is more or less almond-shaped, with its long axis horizontal. Its outer extremity (external canthus) is sharp, and its inner extremity (internal canthus) rounded. At the inner canthus you can see the *caruncle* and the *plica semilunaris*. The latter is merely a fold of conjunctiva, and represents the nictitating membrane found in many lower vertebrates. The caruncle is an island of skin covered by conjunctiva, and contains sebaceous glands; springing from it we can often detect a few soft hairs. Occasionally these hairs are so long and stiff as to be a source of irritation.

You will observe that no lashes are found on either lid for a distance of about 5 mm. from the inner canthus. Where the lashes cease there is a small elevation, the lacrymal papilla, presenting at its apex a small opening, the *punctum lacrymale*.

Through these puncta, as you know, the tears leave the conjunctival sac, and we must therefore note their position and potency when there are symptoms of lacrymal obstruction.

We now test the power of the orbicularis in closing the lids, and of the levator in raising the upper lid.

The *conjunctiva* is a thin, transparent mucous membrane, which we have seen lining the inner surfaces of both eyelids; it is then reflected forwards over the front of the eyeball. The sharp bend it makes in passing from the eyelids to the eyeball is known as the conjunctival cul-de-sac or fornix. Over the sclerotic it is an independent, freely movable structure, supplied by blood vessels running on its deeper surface; these vessels are always distinctly visible through the conjunctiva, and can be made to move along with it, so that they are readily distinguished from more deeply lying vessels firmly attached to the sclerotic. We are thus enabled to differentiate between engorgement of superficial or conjunctival and of deep or episcleral blood vessels. At the corneal border the conjunctiva becomes closely adherent to the deeper tissues, and passes over the cornea as a smooth, thin, non-vascular epithelial covering.

As to the *sclerotic*, I have little to say now. Its most important clinical characters are its colour, its curvature, and its opacity. The colour ought to be uniformly white, except where it is perforated by vessels, where we often find very small, dark brown patches. In old people the sclerotic may be yellowish, and has often yellow prominences upon it near the outer and inner edges of the cornea. Such prominence is called a pinguicula; its colour is due partly to the presence of a yellow colloid material, and partly to the formation of elastic fibres in the conjunctiva at this spot. In children the sclerotic is usually bluish-white, on account of its thinness allowing the deep choroidal pigment to show through. You should observe the curve of the sclerotic; any localized bulging (staphyloma) or patch of discoloration must be noted.

Where the sclerotic joins the cornea we find a fine furrow running all round, called the scleral sulcus; it is due to the difference in the curvature of the two tissues, and to the manner in which they become continuous.

The Cornea.—We notice that it has a glistening surface, that it has a fairly uniform curvature, and that it is transparent. To examine the cornea let your patient sit with his face to the window in a good light. You then proceed to examine for any irregularity of the surface (abrasion, or ulceration, or depressed cicatrix), and for any haziness of the corneal tissue. On looking at this patient's eye we see on it the reflection of the window. The lines of the window-frame are all regularly represented on the corneal surface, showing that there is no

marked irregularity in the curve. Again, when the patient moves his eyes in different directions, so that the reflection appears on every part of the cornea, there is here no interruption or blurring of the image, as there would be had the window-lines fallen on any abraded or deformed surface incapable of reflecting the image truly. This observation of the window-reflex, therefore, is convenient and most important in examining for irregularities of the surface or curvature of the cornea.

Next, we observe that this cornea is transparent, permitting the deeper structures to be seen quite distinctly through it. In the cornea of oldish people we often find an arc or even complete ring of opacity near its circumference, familiar to you as the *arcus senilis*. Any other opacity seen would be pathological, due either to cicatricial tissue or to an existing inflammatory condition. The opacity may be due to changes on the anterior or posterior surfaces (both epithelial), or in the intermediate proper corneal tissue. But to detect the finer opacities of the cornea, to examine them more minutely, and to estimate their depth, we have recourse to what is known as focal illumination. This method of examination may be conducted by daylight, but still better by artificial light in a darkened room. A biconvex lens of about 3 inch focus and 2 to 3 inch diameter, is held in one hand so that the concentrated light falls on the part of the cornea to be examined; the illuminated area is then observed, either with the unaided eye, or magnified by a second lens held in the other hand. The character of the different opacities of the cornea that may thus be seen will be more fully described in the lecture on affections of this tissue.

If we now view the eye from one side it is possible to judge of the amount of curvature of the cornea, and to estimate roughly the depth of the anterior chamber. Such an examination is important to the diagnosis of conical cornea, keratoglobus, glaucoma, and other ocular conditions.

The *Iris* forms a movable curtain suspended in the aqueous chamber and lying in front of the lens. By the action of its muscles it can be made to contract or expand, so as respectively to enlarge or diminish the size of its central opening or *pupil*.

You can see that a zigzag line divides the iris into two unequal parts; an outer or ciliary, and an inner or pupillary portion. With the aid of a magnifying lens you can trace fine raised bands radiating outwards in the ciliary portion, which branch and interlace at intervals, thus leaving depressions; these latter are the mouths of lymphatic crypts. Movements of the iris suck in by or expel from these crypts the aqueous fluid, and in this way the iris is in part nourished; in the case of an iris fixed by adhesions, this lymphatic circulation is interfered with, and the tissue suffers in consequence.

To ascertain the movements of the iris, or—to express it in another way—to examine the activity of the pupil, place your patient with his face to the light. Keeping one eye carefully shaded with one hand, cover and then quickly uncover the other eye with the other hand, and notice the movement of the iris (contraction of the pupil) provoked by the sudden exposure. Now observe that the pupil of the shaded eye also contracts consensually with the other. These movements are spoken of as the direct, and consensual (or indirect) activity of the pupil to light. The iris also moves so as to produce pupillary contraction, in association with convergence or accommodation. In testing this, both eyes must be equally exposed to light, but must not be allowed to look directly at the source of light; the patient is first told to look in the distance, and then at the point of the finger brought to within a few inches of his eyes, when the pupils will be seen to contract. Any failure in briskness, any feebleness or absence of action under either mode of examination must be carefully observed and noted.

The normal pupil is round, regular in outline, and black; any departure from these conditions is suggestive of iritis or of changes in the lens, or of deposits on its anterior capsule. But these subjects will be fully discussed in future lectures.

There should be no inequality in the size of the two pupils when both eyes are equally illuminated.

Intra-ocular tension.—We gauge this tension by trying the elastic resistance offered by the coats of the eyeball to gentle pressure applied from without. It is difficult to accurately describe in words the manner of estimation, but the method commonly used is as follows. The patient is placed facing you and is told to look downwards; you then place both forefingers on one upper eyelid and exert gentle pressure on the globe through the lid; the one forefinger is used to steady the eyeball, while with the other you press the globe gently downwards and backwards for a moment, so as to judge of the resistance that the sclerotic offers to the slight dimpling so produced. This may be repeated several times, till you get a mental estimate of the readiness with which the sclerotic yields, and this estimate is then mentally compared with your previous experience of the tension of the normal globe. It is well always to compare the tension of the two eyes. When the globe is absolutely rigid or quite soft, it does not require experience to be able to say so; but much practice is necessary to appreciate slight departures from the normal, and to estimate the different degrees of increased or diminished tension.

Ocular movements.—The different movements of the eyes are sufficiently examined by getting the patient to look as far as he can successively upwards, downwards, and to each side, and by

then testing his power of converging the eyes to a near object. The extent to which the cornea is covered by the eyelids at the extremity of the movements affords the best means of measuring the power of rotation, and the two eyes must be carefully compared in this respect with one another, and also with our mental standard of proper movement. Fallacies due to swelling, drooping, or malformation of the eyelids must be guarded against. Observe that, in lateral deviation, the external rectus has not such power of burying the cornea at the outer canthus as the internal rectus has at the inner canthus.

Any unsteadiness of the eyeball, whether mere jerking on forced movements, or true nystagmus, should be noted, and also the direction of the abnormal movement—horizontal, vertical, or rotatory.

Bilateral symmetry.—We learn a great deal clinically by observing differences in this respect: for example, by attending to the size of the palpebral apertures, to the relative position and direction of the two eyeballs, and to the size of the pupils. There are many respects in which the two eyes may obviously differ, from congenital abnormality or from disease, which I need not here particularize.

The question of the *relative prominence* of the two globes is one that not infrequently arises. Judging merely by a superficial look, one is apt to be guided too much by the extent of the eyeball exposed by the lids. To estimate relative prominence more accurately, we proceed in this way. The patient is seated on a chair, looking straight forwards, while the observer stands close behind, and tilts the patient's head gently backwards or forwards until he can get the cornea and the eyebrow (or frontal eminence) of one side in the same line; it is now easy, preserving the positions, to judge whether the other cornea is in front or behind the line of its eyebrow. The only remark to be made regarding this observation is that we must be careful to make due allowance for any inequality in the prominence of the two sides of the patient's forehead.

These, then, are the main facts concerning the external examination of the eye, with which one must be familiar before coming to diagnose eye diseases. When you see a case for the first time, there is, as a rule, some marked symptom noticeable, and you do not require to examine first the lids, then the conjunctiva, cornea, iris, etc., in any definite order. You take the prominent symptom or group of symptoms as your starting-point, and examine the parts concerned; you ask questions as to subjective symptoms, and the answers suggest fresh inquiries and further examinations.

(A number of patients were then examined, and the different points of clinical importance demonstrated in each case.)

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 1, 1896.

CLINICAL LECTURE

ON

A CASE OF CARDIAC DISEASE, WITH LIVER ENLARGEMENT,

ILLUSTRATING THE POSSIBLE EFFECTS OF
SYPHILIS ON THE HEART.

Delivered at the Bristol Royal Infirmary, on October 5th,
1895,

By R. SHINGLETON SMITH, M.D., B.Sc., F.R.C.P.,

Honorary Fellow of King's College, London, and Emeritus
Professor of Medicine in University College, Bristol.

GENTLEMEN,—This patient has been under observation for two or three years, with symptoms of heart failure and liver swelling, presumed to be of rheumatic origin. Again and again have the symptoms disappeared under digitalis treatment, until a long continued attack of diarrhoea produced a progressive asthenia terminating in death. Her history is by no means complete, and does not give any account of the origin or the course of syphilitic disease, which is inferred entirely from the post-mortem discovery of advanced albuminoid disease of liver, kidneys, spleen and intestines, and fibroid disease of the heart.

The details, such as were obtained, are as follows:—

Martha P., æt. 38, a single woman, whose occupation was that of a charwoman, but who appears to have led the life of a prostitute, first came under observation several years ago, when she attended as an out-patient with rheumatic symptoms affecting the middle-sized joints, which did not swell, but she perspired freely, and has ever since shown symptoms of heart defect.

She was healthy as a child, had measles at 20, and then continued well till the rheumatic attack of 1890, when her continued illness appears to have commenced. In 1893 she was ill for several months with sickness and diarrhoea; at this time

her breath was short, and she suffered greatly from thirst; she attended as an out-patient under Dr. Watson Williams.

No children—no miscarriage—but always irregular; mother died from internal tumour at 54, father from syncope at 63, one sister died from dropsy at 10, another was supposed to be consumptive.

She was an in-patient under care of Dr. Shaw in January, 1894, when, after prolonged wasting, she noticed that her legs began to swell; this gradually increased and extended upwards, so much so that she was obliged to lie up. On admission there was considerable œdematous swelling at the lumbar and sacral regions, but there was no œdema of the legs; the face and conjunctiva were œdematous. Pulse was 112, small. There was evidence of dilatation of the right ventricle, with a systolic murmur not conducted into the axilla, and the second sound was accentuated in the pulmonary area. Urine had sp. gr. 1009, and contained 0.1 per cent. of albumen.

On Feb. 24th, 1894, she was again admitted, under care of Dr. Prowse, for swelling of abdomen and legs, with shortness of breath, cough, anorexia, vomiting, and wasting. She then had an apex murmur described as mitral systolic, with the apex beat in the nipple line. There was dulness at the bases of both lungs, with much crepitation, and the abdomen was much distended with fluid, the urine contained albumen, the pulse was regular at 74.

From Dec. 13th, 1894, to Jan. 15th, she was again an in-patient for heart symptoms. She gave an account of acute rheumatism five years before and of constant need for medical attendance since; latterly the difficulty in breathing had increased, and she complained of faintness, palpitation, and pains in the legs on the slightest exertion. Several times during the previous year she had been jaundiced, but there had usually been constipation. She became very cyanosed after exertion.

She was found to have anasarca, with icteric tint, and cyanosis, with the area of hepatic dulness much increased and the spleen normal. A systolic

murmur was audible over the pericardium, but there was no definite apex bruit. Pulse was small, frequent and irregular. Urine, *no alb.* Rapid improvement followed digitalis treatment.

She was finally re-admitted as in-patient on August 20th, 1895, when she was found to be thin, weak, and pallid, with a tendency to cyanosis of the lips. She was short of breath, and this was aggravated by the slightest exertion. There had been some swelling of the abdomen and of the legs, and the liver dulness was much in excess, extending to within an inch of the umbilicus. Appetite was poor, tongue was coated, she complained of thirst, with tendency to diarrhoea, and there was albumen in the urine. The heart apex was much displaced, there was a decided systolic apex bruit, and the pulse was small, weak, and irregular at about 84. There was no lung defect; the presence of some dulness in flanks disguised the area of splenic dulness; there was no abnormal temperature, and the red blood cells numbered 1,510,000 per c.c. Treatments with digitalis and bismuth was commenced with the expectation of speedy improvement as on previous occasions, but on September 9th the usual treatment was discontinued in consequence of increasing sickness and weakness; meanwhile she had developed an attack of acute eczema of the hands, and the heart murmur had become almost inaudible. On September 14th the weakness was much intensified by copious diarrhoea, the ascites appeared to be less, but the liver dulness had not diminished, and the albumen in urine persisted.

On September 18th much bloody mucus was passed from the bowel, the tendency to diarrhoea and vomiting continued unabated, the heart murmur at apex was now inaudible, but there was a loud hæmic bruit in the pulmonary area, and the asthenia was increasing.

There was no return of the oedema of legs, and there were no indications of increasing heart difficulty, but the weakness steadily increased, and led to the death of the patient on October 1st, 1895.

The post-mortem notes made by Dr. Theodore Fisher are as follows:—

Body of a short, fairly well-nourished woman, with scars on left groin such as may have resulted from a suppurating bubo, irregularly-shaped deep scars present on the left side of the mouth and

over forehead, also two circular scars on outer side of left knee-joint.

No oedema of legs.

Brain not examined.

Lungs.—Several enlarged caseous glands around bifurcation of trachea. Some mucus in bronchial tubes. Nothing else noteworthy.

Abdomen.—The pelvic viscera were adherent to one another and to the intestines in contact with them. Both Fallopian tubes greatly distended and full of pus.

The mucous membrane of the small intestines exhibited the appearances of lardaceous degeneration, staining deeply with iodine.

Liver much enlarged, weighed 106 oz. The upper surface of the right lobe was deeply fissured by scars running antero-posteriorly and transversely, and in the right lobe near the anterior border was a grey somewhat depressed translucent area about the size of a shilling, which on being cut into presented the appearances of a recent gumma.

The liver on section was pale and fatty in appearance, but on testing with iodine there was seen to be also a considerable amount of lardaceous change.

Spleen, 12½ oz., soft, but although almost diffused, the malpighian bodies readily stained with the iodine solution.

Kidneys.—One large complete horse-shoe shaped structure, the lower ends of each kidney being connected by a broad band. The whole weighed 9¾ oz. The vessels and glomeruli readily stained with iodine, but there was little evidence of fatty change.

The *Heart* was much enlarged, weighing 23½ oz.

The pericardium was universally adherent, but separated with ease except in the neighbourhood of the apex, where at the anterior and posterior surfaces of the left ventricle attempts at separation commenced to tear the heart substance.

Both ventricles were dilated and hypertrophied, the auricles also were dilated. The orifices and valves on both sides of the heart were normal, the tricuspid admitting four fingers, the mitral three. On the inner surface of the left ventricle was a large patch of thickened endocardium: its centre was visible in the outer wall between the attachments of the two papillary muscles, it spread also down to the apex, and to the left irregular-shaped

branches extended over the septum. On feeling the centre of this opaque patch, it was at once evident that muscular tissue was absent, and section showed the wall of the ventricle to be formed of fibrous tissue of about $\frac{1}{8}$ of an inch in thickness, adherent to the pericardium. The area over which the muscular wall was replaced by fibrous tissue measured about $2\frac{1}{2}$ inches in diameter. The thickening of the endocardium was more extensive than the area of destruction of the muscle wall, hence the appearance of opacity did not represent the area of muscular wasting. The fibrous patch behind ended somewhat abruptly in muscular tissue, but in front and below the fibrous tissue shaded off more gradually into the heart wall, the tissue for some distance being half muscular and half fibrous.

The aorta had some raised thickened patches just above the sinuses of Valsalva, and other patches near the commencement of the transverse portion of the arch, but was on the whole healthy.

For further description and sketch of this heart, *vide Bristol Medico-Chirurgical Journal*, December, 1895.

The post-mortem disclosed what was not known during the life of the patient, that her viscera were in an advanced condition of lardaceous degeneration, and that there was fibroid degeneration of the myocardium with old pericarditis, both probably resulting from gummatous infiltration of the heart wall.

Comment on Case.—The first thing which struck an observer in examining this patient was the existence of a *large and tender liver*, and on subsequent occasions the same feature was usually present. On inquiring into the cause of this, the condition of the heart, there being unquestionable mitral leakage, naturally appeared to give a probable explanation. Such painless (or almost so) enlargements of the liver may be due to many well-known causes, such as—

a. Simple hypertrophy of cardiac origin, the liver varying in size like a sponge in accordance with the conditions of the circulation.

b. Leukæmia, when the spleen will also (one case liver 10 lbs. 8 oz., spleen 13 lbs. 8 oz.) be enlarged, and possibly the lymphatic glands as well.

c. Fatty liver—a case given by Fagge in which it weighed 155 oz.

d. Malarial causes.

e. Neoplasms, including painless hydatid growths.

f. Lardaceous liver. In one case given by Wilks it weighed 14 lbs. This is a somewhat unusual form of liver enlargement, and in the presence of the usual history of prolonged suppuration or of syphilis, the possibility of the liver enlargement being of this nature would naturally suggest itself. In this case the history of rheumatism and the presence of the symptoms of mitral disease rendered it probable that the hepatic swelling was due to cardiac congestion, and the idea of lardaceous disease was not therefore entertained. Rheumatism as a cause of cardiac disease is so infinitely more common than syphilis that even the scars on the skin were considered to be of no importance in the etiology of the condition.

Syphilis is little known as a cause of heart disease, and in the presence of a definite history of rheumatism may readily be overlooked during life, perhaps also after death, unless there should be some such clear evidence of syphilis as is afforded by the presence of albuminoid disease.

Dr. James Little ("Resources of the Physician in Management of Chronic Diseases of the Heart"—Fannin & Co., 1895), speaking on the etiology of heart disease between the ages of thirty and sixty, remarks:—

"Lastly, it is well to remember that sometimes, though very rarely, the valvules and the muscular wall of the heart may be the seat of syphilitic disease," and in evidence of this statement he quotes the case of a gentleman whose heart symptoms were relieved by iodide of potassium.

Some better evidence than this therapeutic observation is needed before we can accept the syphilitic origin of peri-, endo- and myocarditis and fibroid heart; but the case now reported gives us very good evidence in favour of syphilis as the essential cause of the fibroid heart, perhaps also as a cause of endocarditis and of pericarditis with its natural sequel, adhesion of pericardium.

As evidence of syphilis we have the presence of scars such as are usually found after the occurrence of constitutional syphilis; the patient's history was one in which syphilis was likely to occur; the patchy and localized condition of the fibrosis in the heart-walls was unlike any other than the result of a localized gummatous infiltration, with subsequent degeneration of muscle; and

lastly, the presence of the characteristic albuminoid disease of liver, spleen and kidneys. The comparative rarity of syphilis as a cause of heart disease is shown by the statistics of Ashton (*Philadelphia Medical News*, June 30th, 1894), who gives an analysis of 1024 cases of chronic valvular disease, with the following results:—

A history of rheumatism in . . .	37.4 p.c.
„ some infectious fever in 10.0 „	
„ alcohol in . . .	18.0 „
„ syphilis in . . .	9.0 „

and in 21 per cent. no cause could be assigned.—*Sajous' Annual*, 1895. Of these cases (9 per cent.) a history of syphilis was obtained, but it is not implied that there was any positive evidence connecting the heart disease with the history of previous syphilis.

The ways in which syphilis is known to affect the heart are various. The gummata are of course the characteristic lesion; these are usually multiple, forming tumours of variable size, and having the usual cellular structure associated with interstitial inflammation; they may exist in the recent and characteristic form, or in various degrees of obsolescence; the caseous, fibrous, or calcified remains of obsolete gummata will generally be sufficiently characteristic. Again a specific arteritis may affect the vessels of the heart walls, and then the results of thrombosis may be superadded to those of myocarditis; hence we may get patches of fibroid induration in association with more or less of muscular atrophy. In both these ways syphilis plays an important rôle in the production of the fibroid heart, the fibroid induration being either the direct result of the degeneration of the gumma or of a more diffuse syphilitic myocarditis. In whichever way the heart walls are first attacked, it is clear that either the endocardium or the pericardium, or both, may be secondarily invaded, and hence we may get a syphilitic endocarditis with valvular defects or a syphilitic pericarditis with adhesion.

In this case it would seem that the heart walls were first invaded by gummatous growths, and that in the secondary processes associated with the degeneration of the growth the pericardium and endocardium became both involved, although we have no history of a definite attack of pericarditis.

Two cases of fibroid degeneration of the myo-

cardium are commented on by Theodore Fisher (*Bristol Medico-Chirurgical Journal*, Dec., 1895); the second of these illustrates the more common variety of fibroid degeneration of the patchy kind associated with disease of the coronary arteries, and he emphasizes the probability that both the patchy kind and the diffuse post-gummatous infiltrations are due to syphilis rather than to a rheumatic myocarditis.

In the museum of the Bristol Royal Infirmary is an excellent specimen, numbered 44 L, of multiple sub-pericardial growths. (*? Gummata*.) The cavities of the heart are unopened; over the ventricles are irregularly scattered white spherical sub-pericardial nodules, varying in size from a pin's head to a pea. Microscopical examination of one of these nodules shows it to consist of a thick capsule of fibrous tissue, continuous with which is a thick barred meshwork of fibrous tissue through the nodule. The spaces so formed are filled with small round cells with well-marked nuclei. No giant cells are visible, nor are there any indications of degeneration. A (?) primary growth is said to have existed in the liver, but no further account has been found.

An important paper on syphilitic lesions of the heart by Dr. Loomis (*Amer. Journ. of Med. Science*, Oct., 1895) points out that “besides gummata, syphilis gives rise to an indurated myocarditis,” and he adds that “it is only possible to infer the origin of these new growths by the antecedent history of the individual, by the presence of constitutional syphilis, and especially gummata in other situations.” He gives the following table to illustrate the different forms of syphilitic disease of the heart:—

I. Gummata.	1. Recent: soft reddish or grey masses. 2. Old: dry yellow cheesy nodules.
II. Fibroid induration.	Localized: well defined masses, large size.
III. Amyloid degeneration.	Diffused: accompanied by inflammation of arteries.
IV. Endarteritis obliterans, often inducing infarctions.	Intermediate form: outer zone of gumma develops into fibrous tissue, cheesy centre, remains as fibroid mass.

An interesting case in which an endocarditis was demonstrated to be of syphilitic origin was reported by O. Israel (*Epitome, B.M.J.*, Oct. 12th, 1895). In this case there was no clinical evidence of

syphilis, but the presence of gummatous changes in the liver, of chronic endometritis, and of gummata in the heart muscle left no room for doubts that the endocarditis originated in syphilitic changes of the heart walls.

The case now reported goes a step further, and gives good ground for the association of pericarditis with gumma of the heart walls. It is not easy to trace the origin of an old pericardial adhesion, but in this case its connection with the fibroid patch is shown by the greater adhesion there than elsewhere, and there is no clinical history of an attack of acute pericarditis; may not the adherent pericardium be the result of a slow and chronic process originating on the surface of a gumma?

This view appears to be the one adopted by Hyde & Montgomery (Saunders, Philadelphia, 1895), in their new text book on Syphilis, where we find the following paragraph:—"Pericarditis is a rare complication of syphilis; it results from gummatous deposits in the fibrous tissue or from implication of the pericardium by the extension thither of a degenerative process originating in neighbouring organs."

A LECTURE

ON

DISEASES OF THE TESTICLE.

Delivered at St. Thomas' Hospital, June 7th, 1895.

By SIR WILLIAM MAC CORMAC, M.A., D.Sc.,
F.R.C.S.,

Consulting Surgeon and Emeritus Lecturer on Clinical
Surgery at the Hospital.

GENTLEMEN,—Tumours of the testicle, the subject upon which I purpose speaking, possess a special interest, on account of the importance of the function exercised by the organ. They may be either of a fluid or solid character, and it will be convenient to consider the two apart. The swellings I intend to invite your attention to to-day are those of a solid nature; these may be subdivided into swellings which arise in consequence of inflammatory changes taking place in the organ—

either chronic, acute, or sub-acute; and secondly, those due to some general infective process, such as the tuberculous or syphilitic, both of which are very frequent—or again, they may be of the nature of a new growth, and it is to new growths that I purpose addressing myself to-day.

They are of two kinds—simple and malignant; and of the latter there are two kinds, one carcinomatous, the other sarcomatous. I would say at once—although the opinion is not held by everyone—that the sarcomatous variety is very much the more common form of new growth met with in this situation; the carcinomatous occurs less frequently. Of course, both these growths are malignant in character, but other growths occur which are not malignant, and it is very important to distinguish the innocent from the malignant tumours. The most common variety of non-malignant growth is that called adenoma of the testicle, of which there are two examples on the table; and there are fibromata and chondromata, very rare forms of tumour to occur in this situation.

In speaking of new growths of the testicle, it is noteworthy that whilst our anatomical knowledge of their structure is much more perfect than formerly, the clinical discrimination of the different forms of new growth in the testicle is as difficult as it ever was; in some cases the difficulty is so great that it is really impossible to discriminate them, to tell what their exact character may be, or indeed to do more than express an opinion as to whether a given tumour is innocent or malignant. After removal it may not be possible to state exactly the character of some of these tumours without microscopical examination, and even with the aid of the most skilled microscopical scrutiny it is sometimes difficult to be quite sure as to the nature of the growth. Some tumours present definite and distinct characters, while others are so mixed up with different elements and with various arrangements of these elements, that to pronounce a decided opinion is almost impossible. This difficulty of course may be explained partly by reason of the fact that purely homogeneous tumours are not the rule. I might almost say that in many instances it is very certain that different elements are present in one form of tumour, the chondromatous, sarcomatous, myxomatous and fibromatous elements may be combined. Some observers go so far as to say that there may be a mix-

ture of sarcoma and carcinoma. Another complication arises from the fact that certain tumours which—so far as anatomical characters go—would, in other parts of the body, be certainly recognized as simple tumours, appear to possess in the testicle a capacity of dissemination which they do not exhibit elsewhere, and must therefore be placed in the malignant category. The marked tendency to dissemination which some of these testicular tumours possess may perhaps be explained by the abundant lymphatic spaces which are found in the organ, furnishing a ready route by which dissemination takes place in the lymphatic system and elsewhere. This is well seen in the case of sarcoma, which, as you know, has not generally the tendency—except at an advanced period of its existence—to attack the lymphatic glands. In the testicle, sarcoma, which is sometimes alveolar in structure, almost invariably invades the lymphatic glands, especially those of the iliac and lumbar regions, and later the lymphatic glands in the groin as well.

I will first speak of carcinoma, which occurs, or is described as occurring, in two forms, one the medullary and the other the scirrhus type. The first of these is certainly the more frequent, the scirrhus variety being so rare that very skilful and trustworthy observers have failed to discover any instance which could really be believed to be an example of this form of cancer in the situation under consideration. At the same time Nepveu has minutely described the characters of this form of cancer from the examination of as many as nine specimens. I believe there is one example in St. Bartholomew's museum, but it is so rare that it may be regarded as a pathological curiosity, if it be not, as Mr. Butlin is inclined to believe, a fibrifying sarcoma; and Mr. Clutton has recently removed a tumour of this kind, which proved on examination to be of a carcinomatous type. It has not yet been put into the museum, therefore I am not able to show it to you. With that exception, all the specimens of malignant new growth in our museum are examples of sarcoma.

My remarks on carcinoma will be devoted to the soft or medullary kind, so named from its external features more or less resembling the soft substance met with in the brain. This drawing represents very well the external appearance of such a cancer. Here, too, is a specimen

which, both in its outward aspect and on section, shows the characteristic features; you will notice the interior soft whitish structure. These cancers are all of the glandular type, and the disease probably originates within the seminiferous tubules of the organ. As I have already said, there is a difference of opinion about the comparative frequency with which carcinoma and sarcoma arise, and I should think it probable that the reticulated, alveolar character of some of the sarcomata which are here met with may have given rise to a wrong impression, and cause cases of what are really sarcomata to be described as carcinomata; in some instances one may, by careful examination, trace the structure which at one place appears alveolar, passing gradually into a true sarcomatous type, in which the cells lie with a certain amount of stroma interposed and in contact with the blood-vessels of the part. At the same time observers of undoubted competence have described cases in which the microscopic structure was strictly that of carcinoma, in which the cells in the meshes of the stroma were of a distinctly epithelial type, and therefore one cannot but hold that these cases were instances of true carcinomata. In most, however, the alveoli of connective tissue are less well marked than in carcinoma, the stroma is more embryonic in type and less abundant.

The carcinoma is spheroidal-celled, and probably springs from the glandular epithelium of the tubules near the centre of the gland, near the rete, and it very rarely, if ever, occurs as a primary growth in the epididymis. As regards the external features of the tumour, it is more or less ovoid or globular, a form it maintains for a considerable time; but it grows rapidly, and presently the albuginea gives way, and the formerly smooth, elastic, uniform surface becomes studded with bumps, swellings, or irregularities, which cause it to have, at different places, a different consistence, and if the growth continue unchecked or uninterfered with, adhesions to the coverings or tunics of the scrotum take place at an early period. This presently ulcerates, and after a time a fungating mass protrudes through the soft parts, formerly named fungus hæmatodes, or bleeding fungus, which is so characteristic of unchecked soft cancer. At a comparatively early period the glands become involved, chiefly those in connection with the iliac arteries, aorta, and

vena cava. The diseased condition may extend up to the diaphragm or even further, and may become metastatic in the mediastinal glands; as a consequence of this you will find interference with the circulation in the lower parts of the body, as well as oedema of the feet, and oftentimes of the legs, in an extreme degree. Fluid will be poured out in the peritoneal cavity, from the pressure on the veins of that cavity, and there may be lung symptoms, from the deposit of the cancerous material there. I have said that at a comparatively early period the lumbar glands are affected, but it is also noteworthy that the superficial lymphatic glands in the groin are affected as well. You, of course, are aware that the main body of the lymphatic drain from the testicle reaches the lumbar glands. It is from that situation that the testicle descends in early foetal life, and the lymphatic channels naturally empty themselves into these glands. But still it is observed that even before the scrotal wall is at all involved the lymphatic glands in the groin may also become enlarged with metastatic deposit. It is somewhat difficult to explain this. There may be a current in the opposite direction from the deep lymphatics to these superficial ones, or there may be, and I believe there is, a lymphatic communication between the testis itself and the superficial glands. However it may be explained, it is a fact that at a period antecedent to the involvement of the scrotal tunics and skin there is very frequently involvement of the inguinal glands. I should have added that not only are the glands involved, but the disease may spread to all parts of the body in a metastatic form, the vertebrae and the other bones being frequently involved. As the disease advances it tends to involve the entire structure of the testicle; the epididymis is completely lost in the substance of the growth, and the cord is enlarged at a comparatively early period. It is a general enlargement of the cord; the arteries that pass down to the growth—notably the spermatic artery—are very much enlarged, and become as large as the radial or tibial arteries; the veins, bringing blood back from the tumour, become engorged, and the other constituents of the cord of course participate in the increase in bulk. This enlargement, by reason of its early occurrence, is of some considerable diagnostic importance.

With regard to the age at which carcinomatous

disease occurs, it may be said to be quite early in comparison with other forms of carcinoma. It is very rare before twenty years of age, so rare that it may almost be excluded prior to that time, although Mr. Butlin records a case as occurring in a child of two, but he qualifies his statement by telling us that he would be almost inclined to regard it as a form of alveolar sarcoma, rather than to assert that it was a unique occurrence of carcinoma at such an early age. Although it may occur at any time after twenty, the general period of life for these tumours to occur is from thirty-five to forty-five, which is somewhat earlier than carcinoma of the breast, for example.

Regarding the structure of the tumour, it is notable that whilst the fibrous skeleton or stroma is distinct, yet it is small in amount as compared with the cells, which are exceedingly abundant. The stroma is but little developed and that of course accounts for the peculiar appearance which the tumour presents, resembling, more or less, brain tissue. With reference to the diagnosis of these cases, I shall refer to it later, when we have said something about the forms of sarcoma which occur in this situation.

It may here be curious to recall how completely some of the great authorities differ on this point of relative frequency. Rindfleisch considers sarcoma of the testicle the most common tumour, and with this view Mr. Butlin, a very good authority in these matters, coincides. Virchow considers sarcoma rare, carcinoma frequent, and in support of this view I may take the occasion to mention that Langhaus, who examined all the preparations which were used for illustrating the second edition of Pitha and Billroth's great work on surgery, verified—so far as the bulk of these preparations go—the statement of Virchow that carcinoma is the more common form of malignant disease of the testicle. There is another point that might be mentioned with regard to the difficulties that occur in making an examination of these cases microscopically, and that is, that the fibres of the stroma of the carcinoma are found infiltrated with embryonic cells, which obscures its character. On the other hand, as the cells in the alveoli are of a distinctly epithelial type in carcinoma, and embryonic in cases of sarcoma, this, with the different arrangement I have mentioned, ought to enable the observer to make out which is which. Nevertheless

there remain cases in which the difficulty is very great, even to the most skilled microscopist.

Sarcoma, in its rate of growth, the pain which it gives, as well as in its outward characters—form, weight, and consistence—closely resembles a soft carcinoma, and if you remove such a tumour and make a section of it you may find the surface presents features precisely similar to those of carcinoma. Short of microscopical examination it is impossible to discriminate one form of growth from the other.

As to the clinical characters, I repeat they are almost identical in the two forms of disease, so that from them alone you are not able to make a diagnosis. The most one is able to do is to say that from the rapid growth, from the pain and the physical characters of the tumour, and from its mode of origin, from the emaciation of the subject, and the implication of the glands if the disease be at all advanced, the tumour in a given case is probably of a malignant nature; but as to which form of malignant disease it is, whether carcinoma or sarcoma, it is impossible to tell.

With regard to sarcoma there are of course different forms. The most common variety—that which is present in nearly every one of the preparations in our museum—is the round-celled; the spindle-celled variety is much less frequent, and less common than either is the mixed-cell sarcoma. In some forms of sarcoma, perhaps in half the cases, cartilage is mixed with the tumour, but this is present almost exclusively in the spindle-celled form. In the round-celled variety cartilage is not found. In some myxomatous changes occur. Cartilage, when present, proves the tumour to be sarcomatous, but its absence neither excludes sarcoma nor proves carcinoma. Sarcoma may be met with at almost any period of life. It is most frequent from birth up to ten years of age, and between thirty and forty years. Both testes may be affected by the round-celled form, the spindle-celled affects one only.

The disease originates in the connective tissue which surrounds the tubuli seminiferi, and the compressed and altered tubules can be discovered in the growth for a long time; and from thence it spreads, involving the whole gland and epididymis, and invading the cord, in which changes, similar to those already described, take place. Contrary to what sarcoma does elsewhere—it affects the

glands at an early period, those in the abdomen and the more superficial as well; also, in about half the cases or more, it produces metastatic deposit in the lungs, liver, and other viscera. The vertebræ, sternum, and skull bones may also be affected.

If I were to describe the external characters of the disease I should merely have to repeat what I have already said with regard to carcinoma. The tumour is either globular or ovoid in shape; it is hard, sometimes very firm, always more or less elastic, sometimes so much so as to convey a deceptive sense of fluctuation; or there may be cysts in it which are more commonly met with in another form of disease occurring in this situation.

With regard to the involvement of the cord, perhaps it is not invaded quite so early as in the case of carcinoma, but it does become involved at an early period, and this should be looked for in these cases by way of a diagnostic sign.

We have to differentiate these two forms of tumour from innocent growth, and for this purpose they must be taken together as distinct from other swellings in this situation. It must be differentiated from a slow-growing cystic fibroma or the very hard, indolent chondroma, from chronic syphilitic sarcocoele, from hæmatocele, also from a hydrocele, which has greatly thickened walls, and contains opaque fluid, or has become very firm and hard from the extravasation of blood into its interior. You may say there should be no great difficulty in making such a distinction, but Dupuytren, one of the greatest French surgeons, excised an old hydrocele, believing it to be a malignant tumour of the testicle, and hæmatoceles have often been removed for a similar reason. However, the want of translucency, the greater weight, the variable consistency, and the absence of any sign of inflammation, together with the constantly increasing bulk, would distinguish it from a chronic inflammation, and also serve to distinguish it from hydrocele; and most especially does this continual increase in size distinguish it from both hydrocele and hæmatocele. Neither of these swellings is likely to alter much in bulk in a given time, while, on the other hand, a new growth—whether sarcoma or carcinoma—constantly and steadily increases in size. Of course in some cases, such as in this preparation, you find a collection of fluid asso-

ciated with the swelling, and masking it, the fluid being either bloody or clear, and its presence will interfere with the diagnosis, possibly causing you to mistake the case for hydrocele.

One would say that in a young subject (for sarcoma may occur frequently up to ten years of age, which carcinoma certainly does not) the possibilities are in favour of sarcoma as distinct from carcinoma, and of course if there is any cartilage to be felt, that would be presumptive evidence that the case was one of sarcoma. Instances where both testes are affected, would afford a presumptive evidence of sarcoma, as carcinoma only affects one testis; but apart from these points the distinction between the two is difficult.

In the early stage, when the tumour is like the one I show you here, presenting a uniform character, smooth in surface, growing but slowly, you can, I think, understand that it may be exceedingly difficult to distinguish between a syphilitic sarcocele such as this other specimen presents, and a sarcoma of the testicle, of which this is an example. They are very much alike in external appearance. It is in an early stage, where there is a painless, smooth tumour the size of a turkey egg, without appreciable involvement of either the cord or the lymphatic glands, that the diagnosis of the presence of a sarcoma rather than a syphilitic sarcocele is very difficult. There may be hydrocele fluid in both. A syphilitic growth is certainly more indolent, more chronic; it grows much more slowly, and the cord is not thickened, which is an important point, or if it be thickened it is only at a late period. Of course if you choose you can try the effect of treatment on these doubtful cases. But in a case where you have a suspicion that the growth is malignant, it is questionable whether it be wise or prudent to resort to what must necessarily be a prolonged administration of the iodide of potassium or mercury, or both, to see if they produce any effect on the disease. During this treatment precious days or weeks are lost, and the period at which a successful operation might be undertaken is perhaps gone for ever, because I need not tell you that in these forms of disease, it is only at a very early period that successful operation is possible. A better plan is to make an exploratory incision, and to be guided by the characters of the growth, rather than to attempt

treatment by medicines. No great harm is done if you make an incision into an inflammatory swelling, while, on the other hand, if the disease be malignant, you give the patient much better chances of recovery.

There is no treatment for either sarcoma or carcinoma short of complete removal, which is of the least avail, and unfortunately the prognosis is bad in both cases, because before the surgeon is called upon to interfere, or before the diagnosis has been arrived at, the deeper glands have often become invaded by the disease, and the operation can only prove effective after a total removal of the diseased parts.

Patients suffering from sarcoma or carcinoma may present a healthy appearance for a long time, and in the early stages of the disease there is nothing whatever in their appearance to suggest that they are stricken with serious disease. So that in a doubtful case probably the best rule to follow is to make an exploratory incision; this will rid you of the liability—which I myself incurred on one occasion—of removing a hæmatocele in mistake for a malignant growth. If you cut into a hæmatocele you will of course remove, or permit to escape, altered blood material, which presents a very dark, grumous and peculiar character; while the new growth when cut into or punctured, will permit the escape of clear, bright fluid blood.

As a rule, secondary deposits will occur often to an enormous extent within six months after the operation of castration, and glandular implication will be found in two-thirds of the cases from six to eighteen months from the first appearance of the tumour. You will judge from that how difficult it is to meet with cases favourable for the operation of castration. At the same time, even though they be unfavourable, operation is not forbidden, because a large mass which acts as a heavy, dragging weight, is removed from the patient, and his anxiety and trouble are thereby much relieved. If not removed, the growth will presently fungate, and large sloughs will form. The removal brings a period—of greater or less duration—of comparative comfort, and when recurrence takes place it is in the internal parts, and death is less distressing than if the original growth were allowed to remain. If you are fortunate enough to get one of these cases

of the spindle-celled variety, which is comparatively chronic in its course, and which has not, so far as you can discover, invaded the glands, there is then a good prospect that permanent recovery may perhaps follow the operation. The cord should be divided as high up as possible so as to be quite sure of being beyond the limit of invaded structures. You should slit open the inguinal canal, and then tie the vessels of the cord close to the internal ring. I had occasion to do this in one of the preparations I have here to show you. The disease had developed in connection with a partially descended testicle, and I slit open the tendon of the external oblique, and was able to reach well beyond the growth. But, as you have seen, the lymphatic glands were invaded previous to the operation, and the patient died six months later in consequence.

CLINICAL LECTURE

ON

PSORIASIS.

Delivered November 30th, 1895, at the Edinburgh Royal Infirmary.

By W. ALLAN JAMIESON, M.D., F.R.C.P.E.,

Physician for Diseases of the Skin, Edinburgh Royal Infirmary, &c.

THE subject of my lecture to-day is one of considerable importance to you, because, on investigation it is found that it is probably one of the most common diseases we encounter here. I refer to psoriasis.

With regard to the frequency with which it occurs in different parts of the world: in Austria we may take from 2 per cent. to 3 per cent.; in America, according to the most recent statistics, about 4 per cent., while here we may take it as being between 7 per cent. and 8 per cent. of all cases. This tends to show that some climatic influence probably, possibly some racial peculiarity, leads to its prevalence in Scotland and England.

Now psoriasis occurs at all ages of life, but it is encountered but seldom in infants. It is met

with, however, in young people—in fact, the large majority of cases occur between the ages of about five and thirty, i.e. in childhood and early adult life.

The primary situation in which we meet with psoriasis are the elbows, the knees, and the scalp. The elbows and knees are the part of the body upon which it is more frequently met with in its earliest onset, but we sometimes meet with it first upon the scalp. The aspect of a patch is that of a reddened portion of skin, bearing over the larger part of its surface more or less of white, silvery scales, and when these scales are scraped away with the curette or the finger-nail, and scraped down to the subjacent surface, you find that there is a slight oozing of blood from numerous punctiform points, and the cause of this will be explained when I come to speak of the pathology of the disease. It rarely itches in its primary situations, and consequently it remains very little noticed by the patient. The patches on the knee or elbow, or even in the hair, may not be observed, because they do not *attract* much attention. It is only when it begins to spread and advance over the surface of the body that the patient begins to be alarmed lest it should appear upon the face. In this way it attracts notice.

The causes which lead to the further extension of the disease are not always clear. Season undoubtedly has an effect, for we find that in spring and autumn the disease takes on an increased vigour. Lowering influences generally also play a part. If the patient is reduced in any way by overwork—over-taxed by hard work beyond his powers—we often find that the disease takes on an increased action.

Pregnancy and lactation are frequent causes of its extension, and one of the exanthemata, scarlet fever, is blamed in many cases for the onset, or for the extension of psoriasis. You often find that after desquamation is completed, psoriasis, which had been latent, or never seen before, begins to develop. Without doubt the hyperactivity of the skin in scarlet fever has something to do with the production of psoriasis.

The first appearances of psoriasis, apart from the situations in which it first manifests itself, are different from what you usually see.

The plate which I show you here illustrates

remarkably well the first appearances in a case of psoriasis, when it is beginning to spread, and also shows that you have a number of small reddish points, varying in size from a pin's head to the size of a pea or larger, rough and covered over, at all events in the larger ones, with scales that have a silvery appearance. To this stage the name of psoriasis punctata has been given, but the name merely indicates the stage of the disease. Gradually the spots grow larger and more distinct, and the silvery appearance becomes well brought out, as you see depicted on the side of the thorax in this plate.

In the next drawing I show you another change. The centre of the patch is beginning to clear up, while the margins extend, and hence circular or half circular forms are produced, and to this stage the term psoriasis annularis has been applied.

Sometimes, however, as is shown in this drawing, instead of clearing up, it extends, and becomes simply larger, and may have a thick coating of mortar-like scales with a red margin surrounding it. Now this red margin is very important, because as long as this is seen the psoriasis is extending, and when the margin disappears or becomes faint round where the scales are, you may be sure that it is in a non-progressive stage. Sometimes you have a certain amount of clearing up in the centre of some of the patches, while in others it remains uniform. Here again we see the red margin indicating increase.

Sometimes, very rarely, however, the scales assume the appearance here shown. This case, I am sorry to say, I have got no history of. It is taken from Neumann's Atlas, and he has given no history of the case. The scales are black, and in this particular case there is a strong growth of hair upon the thorax, and the man was one who had a strong tendency to the development of pigment, and hence probably the change.

This is a more common and interesting case, where you see the scales are brown and very thick and coarse looking, attached firmly to the part, and occurring not infrequently upon the elbows and knees. In these cases you have thick accumulations of hard scales, and it has been termed psoriasis inveterata, because it is accompanied by very severe symptoms. In cases like this you should always suspect an alcoholic tendency. When alcohol is indulged in to too great an extent it is

apt to produce conditions like this—a heaping up and accumulation of scales of the patch.

You may meet with a stage which is different from the preceding. On the outer side of the arm in this drawing the scales are greenish-yellow, and heaped up like a cockle, and this has led Professor McCall Anderson to call it psoriasis rupioides or rupia-form, resembling the rupia which you meet with in syphilitic affections.

The same condition is seen in this plate, but here we have really a syphilitic basis. This is a case described by Jonathan Hutchinson, occurring in a girl about ten or twelve years of age, and in the *Atlas* the plate is wrongly marked as a case of eruption from inherited syphilis. Now Mr. Hutchinson, on careful inquiry, found that it could not have been inherited, because the patient was ten years of age before she showed any symptoms, and it was then found that the mother had suffered from acquired syphilis two or three months before the first symptoms appeared in her daughter, so there is no doubt that the girl acquired it from her mother, and this appearance about three years later is really a commencing tertiary condition. You see that there are peculiar whitish scales on the red surface, but the reddened areola is much broader. The patch shows the arrangement characteristic of syphilis, and the cockle-shaped crusts. It is called by Hutchinson rupia psoriasis.

In the drawing here again I show you an example of psoriasis of the face, and now I shall bring in a patient who illustrates the condition shown in this plate. You will observe on the face of this girl, on the forehead and extending down into her eyebrows, exactly the same condition as is shown in the plate I have just handed round. It occurs also on the head, but mostly on the face. To make sure we have it here on the arms—the elbows and extensor aspects—showing the ordinary appearance of psoriasis in a not very active condition, either standing still or retrogressing. As in this case, the scales sometimes present a yellowish appearance, which depends upon the presence of minute particles of pus between the scales, and cases like this have, to a certain extent, a strumous basis, and are benefited by the administration of cod liver oil.

This plate is a peculiar one. You will notice that there is represented on the back of the patient a number of patches that look like ordinary

psoriasis. At the time the drawing was made, there was a great deal of discussion going on about the supposed parasitic basis of psoriasis. I removed a number of scales from the patient, and found abundant parasitic growth, which I at first thought to be conclusive evidence. However, on examining more carefully, I found that it was the ordinary *microsporon Furfur*, and, on looking into the case, I found that the patient had suffered from *tinea versicolor* on the back previously, and subsequently from psoriasis, which had raised the scales on the ordinary lesions of this other disease.

In this plate I show you three different conditions. The first on the right represents a very curious arrangement of the scales. It occurred on a man who had ordinary psoriasis on various parts of the body, but on the outside of the arm he had a very peculiar arrangement of the scales. They were heaped up in a little round whorl-like arrangement. I have never seen another case like it, and I had a careful drawing taken of the patch. The man was treated by alkaline baths and salicylic vaseline, and afterwards, if I remember rightly, a little tar, and he got completely, and as far as we know, permanently well.

The drawing of the hand, underneath, is a beautiful illustration of psoriasis of the palm. In this case there was also psoriasis of other parts of the body, but on the palm, especially on the base of the thumb, there was a dry, scaly condition of the surface, which, in places, was sharply defined, and in others it faded away. It was, as I have said, associated with psoriasis elsewhere, and there was no doubt that it was a case of psoriasis of the palm, common in psoriasis, but which, as a condition apart from psoriasis elsewhere, or as a local condition alone, is one of the rarest in dermatology.

The last of these drawings illustrates an example of a form of syphilis—a tertiary form—which resembles psoriasis very closely, but you will distinguish it at once, because, on looking closely in, you find that the scaly spots, which are arranged in a horseshoe-like manner, have left behind them a scar. This psoriasis never does, although it may leave pigmentation. Therefore, when you have a condition resembling psoriasis, but leaving a distinct cicatricial condition behind, you may be certain that it is not psoriasis, but most probably is a syphilitic affection.

Sometimes psoriasis affects the nails, and then it attacks the matrix, as a rule, raising it up. Sometimes it affects the lunula, and sometimes it makes the nails rough and striated and ill-coloured.

It does not affect the hair, as a rule, and does not make the patient bald. On the scalp, however, it is occasionally met with in bald people as an erythematous condition, and in general it extends as a well-marked line over the forehead, scaly and reddish, which enables you to diagnose it.

The morbid anatomy of psoriasis is that of an inflammatory condition with a downgrowth of the interpapillary processes into the rete, and with an increase of the papillæ themselves. There is an œdema of the corium with an exudation of leucocytes, enlargement and convolution of the blood vessels, and, in consequence of this inflammatory condition, a disturbance of nutrition, and an imperfect formation of epidermic scales and a heaping up gradually.

As to the nature of it, psoriasis is often said to be hereditary, and you may often trace it in several generations, while on the other hand you meet with many cases in which heredity is absolutely denied.

The disease is sometimes associated with peculiar arthritic complications (*arthritis deformans*), where you have something like rheumatic arthritis—stiffness of the joints and painful affections. This is more common in France than this country, although I have seen a few cases here.

In cases where children are attending school and suffer from psoriasis, they often stand it badly, and you will often have to remove them from school in order to cure them.

It seems that mental exertion in a growing child is unfavourable to the cure of psoriasis.

The diagnosis I do not intend to enter very closely into. In eczema of the head you have generally some moisture, which you never have in psoriasis. In eczema you may have pustulation—never in psoriasis. In eczema of the body you have itching, which does not occur in psoriasis, unless it be very rapidly spreading.

In disseminated ringworm of the head you have a scaly condition resembling psoriasis, but you find in addition broken hairs and the characteristic fungus.

From syphilis I have already given some of the points of diagnosis.

In anæmic persons it is apt to resemble syphilis. The patches have a bright coppery appearance that resembles syphilis, especially the secondary stage, but in syphilis at this stage you have the other secondary manifestations and constitutional symptoms, such as falling out of the hair and glandular enlargements, and there may be some evidence of the primary sore. The situations of psoriasis also help to distinguish it from syphilis. In the former we have the elbows and knees affected, while in the latter, in the secondary stage, these parts are generally avoided; you may have it on the head. Another point which enables you to distinguish it is the history. In psoriasis there is generally a history of former attacks of a similar nature.

Lichen planus is often mistaken for psoriasis, but the small square-shaped, sometimes round, crimson-red papule of lichen planus is quite distinct from the brownish-red, scaly, ill-defined spot of psoriasis.

Psoriasis sometimes ends in exfoliative dermatitis. Instead of remaining localized, it becomes diffuse over the whole body, and you lose the characters of psoriasis in a general redness of the skin, with a constant exfoliation of thin papery scales. This is especially liable to occur in alcoholics, or old people, or patients who are run down.

As regards the nature of psoriasis, further, I may state that it has been supposed by some to be a parasitic affection. As regards that, however, there is but little positive evidence. The parasite, if there be one, has not yet been discovered, and therefore, although it presents in some respects the appearance of a parasitic affection, by spreading from one focus to another, and its ring-shaped arrangement, which is characteristic of some parasitic affections, still, until we absolutely find the parasite, we cannot say that the disease is of such a nature.

The most important point now remaining for consideration is the treatment.

At the present moment we are possessed of no definite means of guaranteeing a permanent cure, but we can cause a disappearance of the disease, at least for a time. Sometimes, however, a cure does take place. Sometimes it disappears, only to

re-appear after a longer or shorter time. In this case before you, you have a good example of the commencement of the disease—small reddish spots, rather ill-defined at the margins, slightly scaly, and tending to disappear to a large extent under pressure. This has been treated by washing with a super-fatted potash soap, and after by the application of salicylic vaseline, and a certain amount of improvement has taken place.

In the first place, as regards treatment, it is most important to bring our remedies into as close a relation to the rete mucosum as possible, and primarily, therefore, we must bring about the removal of the scales. Well, you may do this in various ways, such as oil-packing and alkaline baths, which latter are very valuable. We also may make use of the valuable properties of salicylic acid in causing the separation of abnormal horny accumulations from the skin. We may employ it in the strength of 5 per cent. to 10 per cent. in vaseline, and have it used after a bath. This method is being employed in this case, which a little while ago was very extensively marked, and now, under the use of alkaline baths, followed by the salicylic vaseline, as above, the skin has become smooth. Although we can still define the patches, yet they are not so prominent as before treatment. There is still a certain amount of redness and roughness of the surface, but at the same time the disease is already showing signs of improvement.

In the case I now show you the same process of treatment was commenced with as in the other, only when we got the scales removed we then employed chrysarobinum, which is one of the most valuable remedies we possess for this disease, but it must be used properly and carefully—in the proper kind of case and at an appropriate time. It is of no use employing it if the disease is in a process of advance or increase, or if there be much inflammatory condition of the skin, or if the part feel hot. In such conditions it is out of place. But if you are sure that it has come to a standstill, if the marginal areola has almost or completely disappeared, and the patient tells you that no new spots have appeared recently elsewhere on his person, and that all itching has ceased, then is the time so to employ it. You must try it first with alkaline baths, or with soap (green soap or soft soap) and water and salicylic vaseline, to get thoroughly rid of all the scales, because to make

use of an active remedy, and an active one like chrysarobinum, for psoriasis covered with scales is simply wasting a somewhat expensive remedy, and bringing a valuable one into ridicule. Get the surface entirely free from scales, then chrysarobinum in the form of an ointment of the following strength may be employed :—

Chrysarobinum	...	gr. xxv.
Salicylic acid	...	gr. x.
Ichthyol	...	℥. xxv.
Green soap	...	℥ss—3j.
Vaseline	...	ad 5j.

The ointment should be rubbed in carefully every day, after washing the part with soap. After a little while the patches get paler, and this is most marked at the edges of the patch and less so in the centre, while round the patch on the previously unaffected skin an erythema begins to make its appearance, giving the skin a purplish red colour. This, however, does not contraindicate the further use of the ointment. After a little the pale spots become less pale, and approximate more and more to the dark purplish brown colour which the skin now assumes, and we still persevere with the remedy. As it goes on, the patches approach nearer and nearer to the normal in appearance, but still show reddish spots, which indicate that the application must still be continued.

Gradually this goes too, and finally the skin is left smooth, but darker. Now you may employ a little calamine lotion. The skin desquamates where the erythema had been, and finally the case is so far cured; but we cannot make sure that the disease will not recur.

Other remedies are sometimes employed to remove the scales quickly. The following formula is that of a very useful ointment for this purpose :—

Ammonium carbonate	...	x. parts.
Lanoline	...	xxv. „
Cold cream	...	50 „

This forms an application which rapidly removes the scales, and paves the way for further treatment.

Some case do not get well under chrysarobinum, but, on the contrary, get worse. In these cases you must stop the application. They often do well with tar in one of its various combinations.

The tar may be used as an ointment, emulsified with tincture of quillaia and glycerine of

starch, in varying proportions from $\frac{1}{4}$ per cent. to 50 per cent., or it may be employed simply as an emulsion, with tincture of quillaia, in the proportion of one ounce to the pint.

Pyrogallic acid may be used as an ointment, but it has one great disadvantage, namely, that if it be used over very extensive surfaces it is apt to be absorbed to a dangerous extent and to produce hæmoglobinuria, with a destruction of the red blood corpuscles. It is best suited for application to the head. An ointment may be used consisting of :—

Pyrogallic acid	...	gr. xxv.
Ichthyol	...	℥. xxv.
Salicylic acid	...	gr. x.
Vaseline	...	ad 5j.

The application of this ointment should be preceded by washing the part with soap.

These are some of the more important remedies at our disposal for external administration. Are there any by the internal exhibition of which we may hope to modify the progress of the disease? Arsenic has this reputation, and there can be no doubt that some cases are kept under control by the administration of this drug. It is often of value when its administration is combined with the application of external measures; but while it does control psoriasis, it is very seldom that it cures the disease.

M. Besnier states that cases constantly present themselves at the St. Louis Hospital at Paris that have taken arsenic for very long periods—in some cases as long as twenty or thirty years—and are not cured. But looking around the profession generally, there are few practitioners who have not had some cases that were benefited at one time or another by the use of arsenic. The greatest amount of good is obtained in constitutional cases and in psoriasis of the head, and in the treatment of first attacks. It seems to lose its effects and influence after administration two or three times. I use the liquor arsenici hydrochloricus in preference to Fowler's solution, because the latter contains a certain amount of the compound tincture of lavender, which imparts a nauseating taste to it. In addition, the acid liquor is useful to combine with iron or nitro-hydrochloric acid, and when pyrogallic acid is being employed, this latter is useful to counteract its objectionable effects.

Iodide of potassium in large doses has been recommended by Prof. Haslund, and in some cases may have done good, but the doses are so large and the undesirable effects so great that I cannot recommend it.

Dr. Crocker has lately recommended the use of salicylate of soda, and here, if you give it, you must use that prepared from the oil of Wintergreen, and not that which is artificially prepared. It may be useful in some cases, but I have not found it so far of great value.

Thyroid extract has been advocated by Dr. Byrom Bramwell, and in some cases with remarkable and strikingly good results. We are not yet quite certain, however, as to the exact cases in which it is to be employed, and looking upon it as a remedy that is potent, I could not advise you to prescribe it to your patients unless you can have them under careful constant observation—under, in fact, a trained nurse or in hospital—because the doses, it would appear, are large, and, apart from any unpleasant effects which it may produce, such as palpitation, headache and giddiness, I find there is a certain amount of danger attending the administration of it in large doses. Therefore, at the present moment, that it is useful in some cases is all that we can say.

Can we in any way influence the recurrence of the attacks? In the present state of our knowledge, all that we can say is, as in influenza, "Keep the general health up to the standard." Salt water baths are of value, and we must see to the correction of anything wrong in the system generally.

The rheumatic and gouty tendencies must be counteracted and modified by the use of the alkalis and colchicum. We must also see that the patient takes a proper supply of fresh vegetables, and that alcohol is taken only in the proper physiological quantities.

In very obstinate vomiting in cholera infantum, Dr. Rehfeld has seen most excellent results from

Emulsio Sem. Papav. 100 grams.

Cocain hydrochlor. '01 gram.

Sig. One teaspoonful to be given every hour.

Therap. Monats.

REVIEWS.

Annual of the Universal Medical Sciences.

Edited by CHARLES E. SAJOUS, M.D.

F. Rebman, London. Five Vols.

Published at £3 5s.

The Editor apologises for the lateness of the appearance of the 1895 issue: considering the enormous mass of information contained in these five volumes, we consider that no apology is due, in fact, that praise is rather due that the work should appear at all during the current year.

This, the eighth year of production of the work, is the third in which we have been entrusted with the task of—we can hardly say reviewing it, for such a task is impossible in a publication of the character of the *Clinical Journal*—bringing it before the notice of our readers.

We may say at once that of all the literature of the past year which has appeared bearing on professional knowledge in its widest aspects, that which has not been read, analysed, and reproduced in concentrated form, is hardly worth reading or reproduction, and we feel once more constrained to say that if a medical man will forsake all other books and cling only to these volumes, he will be fully posted up in the latest advances in all directions.

Under Dr. Sajous's supreme editorial command no less than 85 associate editors have worked to produce this most perfect epitome of progress. Not content with this army of assistants, he has enlisted 139 corresponding editors to help in collecting material from every quarter of the globe—England, Scotland, Ireland, France, Austria, Belgium, Denmark, Germany, Australia, Brazil, India, Mexico, Canada, Cyprus, Spain, Persia, China, Argentina, Switzerland, Venezuela, Natal, Cuba, Russia, Turkey, Poland, Italy, Sweden, Honduras, Bermuda, Nicaragua, Portugal, Japan, etc. All have one or more editors and corresponding editors, so that the term Universal is amply justified.

Separate indices to each volume, and a general one in Vol. V., make the work unquestionably the completest and most satisfactory of the numberless publications professing to give an account of the progress of Medicine year by year.

For a table of the actual contents, we refer our readers to our issue for November 21st, 1894, where they are set out in reviewing the 1894 production. Here we can only repeat the words of that Review, and wish it all the success it deserves.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers. Sir I. Pitman & Sons, London.

Price 6d.

This publication of the Society of Medical Phonographers maintains the excellent standard of previous numbers. In the issue for October Mr. Treves, in a pithy, practical article on abdominal section in private practice, lays stress upon the importance of keeping aseptic the wound, and the operator's hands, instruments, and utensils; on the advisability of not sweeping the room, which is to be left without a fire, and made as much like a ward as possible by the removal of carpets and all but necessary furniture. Gas, followed by ether, should be used for anæsthesia, and the patient should remain strictly upon the back for three weeks after the operation. In the after-treatment ice is forbidden, but a teaspoonful of boiling water may be given seven hours after the operation, and the same with a little milk on the next day. On the third day beef tea and jelly are allowed; on the fourth day the bowels must be moved, and on the fifth a little solid food may be given; and on the eighth day the stitches are to be removed. Dr. Fletcher Beach ably discusses the causation and treatment of sporadic cretinism; phthisis, inherited mental or neurotic disease, fright during the mother's pregnancy, and prolonged labour being all given as causes in different cases. The treatment by thyroid extract is very satisfactory. Notes of Mr. Davies-Colley's clinical teaching, and a reference to the death of the late Dr. Carslaw of Glasgow, one of the increasing number of practitioners who use shorthand in their daily work, complete the number.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers. Sir I. Pitman & Sons, London.

To the issue for November Dr. Gowers contributes the third of his series of problems in diagnosis, a case of headache, followed by swelling of the eyelids, protrusion of the eyeball, and general convulsions, serving as the text for the discussion of what Dr. Gowers deems an unknown disease—inflammation of the fibrous and cellular contents of the orbit due to rheumatism. The article

deserves careful perusal. Dr. Neil, of the Warneford Asylum, narrates three cases of melancholia, all of which recovered after symptoms had lasted 11, 9½, and 7 years respectively; and in all the cases features, said to be of bad omen, were present. Dr. Martin's note on delayed desquamation after scarlet fever completes an excellent number.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers. Sir I. Pitman & Sons, London.

The philosophic and the practical are happily combined in the number for December. Mr. Roger Williams's article on the pathogenesis of cancer supplies the former. He declares himself an opponent of the germ theory of the origin of cancer, and believes that neoplasms arise from the abnormal play alone of forces within the body. Hyperplasia, and not inflammation, he regards as the starting point of all neoplasms, and there are no cancer or tumour germs other than the existing cells of the body. When the proper amount of structure has been attained by the growth of a part, the living forces of the body preserve an equilibrium which prevents further development. When that equilibrium is lost cell growths begin afresh; and whilst in the higher animals tumours and cancers result, in plants and the lower animals abnormal germination, known as agamo-genesis, takes place. Dr. Fauscot, in an article on the administration of gas and ether, speaks well of Clover's apparatus and methylated ether. The chief danger is asphyxia, and it is possible to anæsthetise the patient in two minutes, though haste in administration is mentioned only to be condemned. The writer of this notice has himself taken ether alone three times, and uses it in hospital as well as in private practice. He found no difficulty in taking it, it had no bad effects, and he believes that the difficulties of administration are, in ninety cases out of a hundred, the fault of the administrator; for there is an art in giving ether as there is in extracting a cataract. Dr. Huggard of Davos, in the beginning of what promises to be an interesting article on the selection of cases of phthisis for climatic treatment, points out that the general health of the patient, rather than the physical signs in the lungs, should guide the physician in sending cases for climatic treatment.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 8, 1896.

CLINICAL LECTURE ON GASTRIC ULCER.

Delivered at the Westminster Hospital, on December 11th, 1895.

By F. de HAVILLAND HALL, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—In looking round the wards for a suitable case to serve as a text for a clinical lecture, my eyes fell upon two patients in adjoining beds, who were admitted with the symptoms of gastric ulcer, and it occurred to me that I might occupy your time very usefully by directing your attention to some of the points of interest in connection with ulcer of the stomach.

In discussing the etiology of gastric ulcer, attention is at once attracted to the disproportionate number of cases which occur among females as compared with males; or, rather, I should say the greater frequency with which this disease is diagnosed clinically in females than in males. When, however, we enter the post-mortem room we find no evidence in favour of a marked preponderance of cases in females; the statistics of the Westminster Hospital during the ten years ending December 31st, 1894, show that five cases of gastric ulcer were met with in males and six in females. These figures, though few in number, are sufficient to prove that it is extremely unlikely that fatal cases of gastric ulcer occur with much greater frequency in females than in males. Fagge and Pye-Smith, on the contrary, state that out of 101 fatal cases extracted from the post-mortem records of Guy's Hospital there were fifty-nine in men and only forty-two in women. As it seems improbable that gastric ulceration should be attended with a much greater mortality in males than in females, the inevitable deduction which must be drawn from the comparative frequency of gastric ulcer among males and females clinically and in the post-mortem room respectively, is that

the diagnosis in many of the cases occurring in females rests upon an insufficient basis, and that the severer forms of dyspepsia attended with gastralgic attacks, to which females seem particularly subject, are liable to be confounded with ulceration of the stomach.

In a typical case, in which the three symptoms of pain after food, vomiting, and hæmatemesis occur in a young person, the diagnosis of gastric ulcer is easily made, but cases are frequently met with in which the greatest diagnostic acumen is required to arrive at anything like a definite conclusion.

The first difficulty that has to be met is to distinguish cases of hæmatemesis due to cirrhosis of the liver from those due to ulcer. This difficulty arose in the case of E. W., a woman aged 37, who was under my care in Tillard last October. The history she gave of herself was that her appetite was poor, that her food was taken at irregular intervals, but that she only drank a pint of stout daily, and whisky occasionally at night. During the past three or four years she had suffered from retching immediately on rising in the morning. This fact, together with the existence of a somewhat diminished area of liver dulness, led me to regard the hæmatemesis in her case as being due to cirrhosis of the liver rather than gastric ulcer. The difficulty practically only arises in the cases of hæmatemesis in quite the early stage of cirrhosis. As a rule, by the time that hæmatemesis occurs the other symptoms of cirrhosis are so well marked that the diagnosis is easy.

In some cases of ulcer of the duodenum, hæmorrhage may take place into the stomach as well as into the intestine, as occurred in W. J., a man aged 31, at present in Burdett Ward. In these cases the hæmorrhage may come on without any previous evidence of gastric disturbance. The differential diagnosis is exceedingly difficult; fortunately for the patient it is of no moment, as the treatment is precisely the same for both conditions.

Another difficulty is to distinguish between ulcer and cancer of the stomach. In well marked

examples of these diseases the diagnosis is usually readily made, but cases occur in which it is almost impossible to give a definite opinion, and it is only time which will clear up the diagnosis. As an example of this difficulty I would cite the case of a lady of 47, whom I saw in consultation. She was subject to what she called bilious attacks; there was no suspicion of alcoholism, and she had had an attack of hæmatemesis, bringing up about half a pint of blood; since then she had suffered from constant vomiting. On examination, there was a sense of resistance in the umbilical region. On inquiring into the family history, I found that a sister and two aunts had died of cancer. In this case I had considerable hesitation in arriving at a conclusion. It is rare in cancer for so much blood to be vomited, and the symptoms had come on more suddenly than is usually the case, but the feeling of resistance in the umbilical region, and the family history, pointed strongly to cancer. The patient, however, gradually improved, so that it must be assumed that the hæmorrhage came from a gastric ulcer.

But the difficulty, I take it, which most often confronts us, is to distinguish between cases of neurotic dyspepsia and gastric ulcer. This is, I believe, the explanation of the apparent clinical preponderance of cases of gastric ulcer in the female sex as compared with the male, notwithstanding, as I have already pointed out, that deaths from gastric ulcer are pretty equally divided between the two sexes. And here I must frankly confess that there are no absolute criteria by which you may be able to distinguish between the two conditions. Even hæmatemesis, which by some authorities is regarded as of decisive moment in making the diagnosis in favour of gastric ulcer, has been known to occur when at the post-mortem no breach of surface in the mucous membrane of the stomach has been detected. The presence of superficial tenderness as opposed to deep tenderness, the occurrence of pain independently of the taking of food, or the fact that pain is relieved by taking food, and the existence of neuralgia affecting other parts of the body, suggest a neurosis of the stomach rather than gastric ulcer.

As regards the terminations of gastric ulcer, reference to the post-mortem books shows that of the thirteen cases which have come to an autopsy, in five death was directly due to loss of blood, in

three death was due to perforation, in two death was due to exhaustion from constant vomiting. In two cases the ulcers had healed, death being brought about by other causes, and in one case the ulcer was found at the post-mortem on a surgical case, and there are no particulars as to the cause of death.

These figures, though few in number, sufficiently indicate the dangers to be feared in cases of gastric ulcer; but they are not enough to furnish any data for the comparative frequency of the cause of death. For instance, the number of cases of death from hæmorrhage in comparison with those from perforation is excessive. Wilson Fox states that "under the most favourable estimates the danger of death from this cause (*i.e.* perforation) is considerably greater than that from hæmorrhage," and in the *post-mortem* records of Guy's Hospital, out of thirty-four cases death was traceable directly to hæmorrhage in eleven cases, and to perforation in seventeen. In only three of these thirty-four cases was death due to gradual exhaustion.

Treatment.—For the sake of convenience I am accustomed to divide the cases of gastric ulceration into five groups when considering the question of treatment. As you, gentlemen, are attending the out-patient department as well as the wards of this hospital I may say that Group I. you will see in the former place, and the remaining four groups for the most part in the wards. Under Group I. I include all those cases of suspected gastric ulcer in which the symptoms are not so severe as to confine the patient to bed. Under Group II. I place cases of such severity as to require the patient to be in bed. Group III. cases in which copious hæmatemesis has occurred, or in which symptoms suggestive of perforation have been noted. Under Group IV. are included cases in which perforation has actually occurred; and, lastly, under Group V. are placed cases of dilatation of the stomach, secondary to the cicatrization of an ulcer in the neighbourhood of the pylorus.

As regards cases belonging to Group I., regulation of diet is the all-important element in treatment. During the nineteen years I was physician to out-patients I had naturally a large experience in the treatment of these cases, and I found that the patients were very ready to adopt the diet I advised. In the great majority of cases recovery took place, in a few the symptoms became so severe as to

necessitate admission into the hospital ; but in no instance had I occasion to suspect that a fatal result, either from perforation or hæmorrhage, occurred while the patient was under treatment in the out-patient department ; this may, however, have happened without my knowledge. The dietetic treatment I am accustomed to recommend in these cases is that the patient should take food every two hours ; about five ounces of milk, beef-tea, mutton, chicken, or veal broth is the quantity I order. Three out of every four meals, or at least two out of every three should consist of milk. The milk should be boiled, and given either warm or cold, as the patient prefers. If milk alone disagrees it may be given mixed with lime-water or soda-water. In private practice, if pure milk is not digested, it can be peptonized, but in the class of patients attending in the out-patient department this is not practicable ; so that if milk cannot be digested the patient should be admitted into the hospital. The same rule holds good if the patient cannot take five ounces of fluid at a time. Some patients get on better if Bengers' food or one of the other partially digested foods is mixed with the milk. I have found that Koumiss can sometimes be taken when milk is rejected, but Koumiss is not an article one can order for out-patients. As regards the medicinal plan of treatment, I usually order our *haustus bismuthi et sodii*, which consists, as you know, of ten grains each of carbonate of bismuth and bicarbonate of sodium in an ounce of peppermint water. The bismuth is better given without any mucilage to suspend it. I have found that this mixture given on an empty stomach three times a day, or every four hours, according to the urgency of the symptoms, relieves pain and checks acidity and the tendency to vomiting. If the bismuth mixture fails, I order the *haustus acidi hydrocyanici et sodii*, which contains ten grains each of the bicarbonate of sodium and the carbonate of magnesium, three minims of dilute hydrocyanic acid, and twenty minims of compound tincture of cardamoms in an ounce of dilute chloroform water. If pain is a prominent symptom, five minims of liquor opii sedativus, or the same amount of liquor morphinæ bimeconatis may be added to each dose of the medicine, and cautiously increased if necessary. As out-patients cannot at the outside be seen oftener than twice a week, it is particularly important that no more opium or other powerful

drug should be ordered than is demanded by the necessities of the case. In cases in which the pain is of an excruciating character, the application of a mustard-leaf or of a blister over the painful spot will often give much relief. As many patients suffering from gastric ulcer are costive, it may be necessary to order an aperient ; for this purpose I have generally employed the *pulvis sodii compositus* of our Hospital pharmacopæia. This contains a drachm and a half of sulphate of sodium, fifteen grains of bicarbonate of sodium, and five grains of chloride of sodium. A teaspoonful of this powder dissolved in a tumblerful of hot water and taken before breakfast makes an efficient aperient. In private practice Carlsbad salts or one of the mineral waters may be employed. I am inclined to attach much importance to the beneficial action of the hot alkaline solution on the mucous membrane of the stomach. If the saline is not sufficient, one of the fluid preparations of cascara may be ordered in addition at night. The formula we use in the Hospital answers well. It consists of half a drachm of liquid extract of cascara, liquid extract of liquorice and glycerine respectively, in an ounce of water. Should flatulence be a troublesome symptom, soda-mint tabloids will usually be found very serviceable. If, under the plan of treatment I have sketched above, the patient begins to improve, *i.e.* when sickness and pain cease, then the diet may be cautiously increased—some light pudding may be ordered, or a lightly-boiled egg with a thin slice of bread and butter, until at last ordinary solid food is taken. I always defer allowing tea as long as possible. If the patient is anæmic, as is usually the case, I order two or three minims of liquor arsenicalis in combination with ten grains of the ammonio-citrate of iron as soon as the patient is able to take meat.

I have entered somewhat fully into the minutiae of treatment, as I consider it one of the advantages of clinical lectures that the lecturer, not being trammelled by any fixed rules, can devote as much time as he thinks necessary for any subject, and can treat it more in detail than is possible in a systematic course of lectures.

I will now turn to the treatment of Group II., cases in which, though the symptoms are sufficiently severe to require admission into the hospital and confinement to bed, nevertheless, the patients can

be fed by the mouth. Under this head will come cases which have not improved in the out-patient department, as well as cases coming on acutely. The bulk of the patients admitted into the hospital with the symptoms of gastric ulcer belong to this group. The treatment is precisely the same as for cases in Group I., with the exception that the patient is kept in bed. Rest in the recumbent position has doubtless a most salutary effect on the cure of a gastric ulcer, but I think that the regularity with which the patient is fed in the hospital, and the certainty that he only gets what is ordered, also greatly assist in accounting for the rapid manner in which these patients usually respond to treatment in the wards. If, on admission, the symptoms are urgent, *i.e.*, if there be frequent vomiting and much pain after taking food, the patient is ordered a tablespoonful of milk every half hour; if a tablespoonful is not retained, the amount is reduced to a dessert-spoonful, or even a teaspoonful. If, however, even this small quantity is rejected, the case comes under the head of Group III. If, on the other hand, the stomach becomes more tolerant, the quantity of milk is gradually increased and the frequency of administration lessened. The subsequent treatment is the same as for Group I.

Under Group III. are included those cases in which profuse hæmatemesis has occurred just before admission—as in a woman aged 33, who was admitted into Tillard Ward on February 9th, 1895, with the history that she had vomited about a pint of blood four days previously—or cases in which hæmatemesis occurs while the patient is in the hospital, and lastly, cases in which the symptoms point to the possibility of a small escape of the contents of the stomach, limited, however, by adhesions. This was my diagnosis in the patient A. I., a woman of 26 years of age, who was admitted on November 5th, and whose case suggested this clinical lecture. The line of treatment you saw me adopt in this patient was, at all events, justified by the result, so that I do not think that I can do better than briefly recapitulate it. When I saw the patient the night of her admission she was in acute pain, there was excessive tenderness over the abdomen, with an elevated temperature. The first thing to be done was to relieve the pain, and almost shock, from which she was suffering.

These symptoms I thought would be best met

by a subcutaneous injection of morphia, and warmth to the abdomen. Accordingly, I ordered $\frac{1}{2}$ grain of morphia to be injected at once, and gave directions that fomentations should be applied to the abdomen and frequently renewed. To prevent straining I ordered the urine to be drawn off by catheter, three times in the twenty-four hours. Lastly, I had to make provision for the maintenance of the patient's nutrition; this I did by directing a peptonized beef suppository to be introduced every three hours and the rectum to be washed out with half a pint of warm water once in twenty-four hours. By the mouth the patient was ordered nothing except small pieces of ice to suck occasionally, to relieve thirst. This line of treatment was continued for four days, then the patient was ordered half an ounce of equal parts of milk and soda-water every hour, and suppositories every four hours. Next day the amount of milk and soda-water was doubled, and the suppositories were discontinued. Two days later the patient was ordered an ounce of milk with an ounce of soda-water every hour, and the next day she had five ounces every two hours, and the day following she was ordered beef-tea and a little thin bread and butter; two days later she was ordered fish; but she was not allowed to get up until three weeks after admission.

The question of the treatment of hæmatemesis resulting from gastric ulcer must be considered here. If moderate in amount I prefer to treat the patient on exactly the same lines as the patient whose case we have just considered, and the results that have attended this plan of treatment in the past encourage me to persevere with it in the future: should, however, the hæmorrhage be of an alarming character, or be repeated from time to time, astringents may be tried. I prefer ergot subcutaneously. three to ten minims of the official injection of ergotin can be used. Some authorities recommend sclerotic acid in preference to ergotin, as it causes no inflammation at the seat of puncture. The dose for hypodermic injection is half a grain. By the mouth three or four grains of acetate of lead in solution with ten minims of dilute acetic acid and five minims of tincture of opium, may be given every two or three hours for six doses. This is a better plan of administering lead than the *pilula plumbi c. opio* of the Pharmacopœia. Gallic acid and dilute sulphuric

acid have also been recommended, and found useful.

In Group IV. are included cases of gastric ulcer in which perforation is diagnosed to have occurred. In these cases the sooner surgical aid is sought the better for the patient. In the meantime nothing should be given by the mouth. If the collapse is marked ether may be injected subcutaneously, and an enema of beef-tea and brandy administered. If pain be excessive, a subcutaneous injection of morphia should be given. For the subsequent treatment I must refer you to my surgical colleagues.

Lastly we come to Group V., which embraces those cases in which the ulcer in cicatrizing has caused pyloric obstruction, and given rise to dilatation of the stomach. The treatment of these cases is, at the best, not a very satisfactory proceeding. In the first place the diet must be carefully regulated. It should consist, as far as possible, of meat carefully masticated, poultry, game, fish, clear soup, milk, and eggs. Farinaceous and saccharine articles are liable to undergo fermentation in the stomach, and cause troublesome flatulence; they should therefore be avoided. In the milder forms of this complaint I have had good results from the administration of the following mixture three times a day before meals:—

R Sodii bicarb., gr. x.
Sodii sulphocarb. gr. x.
Tr. nucis vom., ℥ x.
Tr. chlorof. co., ℥ x.
Aquam, ad ʒ j.
Misce ft. haustus.

In the severer forms the only treatment likely to be of any avail is the systematic washing out of the stomach. This is best effected by means of the syphon plan. A soft œsophageal tube is connected by means of a piece of glass tubing with a rubber tube, to the end of which a funnel is attached. The œsophageal tube is passed into the stomach, and warm water, suitably medicated, is slowly poured into the funnel, when about half a pint has thus been introduced into the stomach, the tube is compressed by the fingers and lowered, pressure is then removed and syphon action allowed to come into play. I have usually employed a drachm of bicarbonate of sodium to the pint of water; some authorities recommend very dilute solutions of boric or salicylic acid. When the patient has become

accustomed to this plan of treatment, a larger quantity of fluid may be introduced into the stomach, and the process may be repeated until the fluid comes away from the stomach quite clear. The patient can even be instructed how to wash out his stomach for himself. I cannot insist too forcibly upon the benefit which usually accrues from the systematic carrying out of this plan of treatment, coupled with a carefully regulated dietary.

In conclusion, I must thank my clerks, Messrs. Pierre and Mackinley, for the notes of the cases on which this paper is based.

TWO LECTURES ON THE GENERAL MANAGEMENT OF LABOUR AND CHILD-BED.

LECTURE I.

Delivered at the Middlesex Hospital, on November 6th, 1895.

By ROBERT BOXALL, M.D., M.R.C.P.,

Assistant Obstetric Physician and Lecturer on Practical
Midwifery at the Hospital.

GENTLEMEN,—To-day it is my intention to speak to you of the general management of labour cases, and next time we meet I will deal with the management of child-bed.

The Lying-in Chamber.—First as to the lying-in room. The doctor is, of course, frequently consulted as to which is the most serviceable room to select as the lying-in chamber. In deciding, there are several points to look to. It is all-important to avoid the dangers which plumbers may have placed in the way. For instance, on several occasions it has happened that a concealed water-closet has been constructed actually in the patient's bedroom, without efficient trapping or ventilation, with the result that foul air has been diffused through the room. This is a serious matter in any chamber, but in a lying-in room the mischief is intensified; not that foul air has any direct influence in producing so-called puerperal fever, that is to say, regarding it in the light of puerperal

septicæmia, but at the very least it must have a potent effect in depressing the vital powers of the patient at a time when they are severely taxed. The room should, if possible, be on the south side of the house, sunny and cheerful; and, in London especially, it is advisable to select a room removed from the street, to ensure quietude. The room should be kept at an uniform temperature— 60° to 65° —and the air should be changed frequently. To assist this, it is advisable, even in warm weather, to have a fire in the grate; and even if a fire be dispensed with, to keep the damper open, for the chimney serves as an effective air-shaft. Finally, except when the patient is exposed, or the child is being washed, a window should be kept open a little. This can be easily done without causing a draught. For instance, by raising the window a little, and placing a piece of wood under the bottom sash and shutting the window on it, an opening is left in the middle, through which air enters only in an upward direction. If there be Venetian blinds, these can be so arranged as to direct the incoming air upward. Draughts can be warded off by placing a screen in a suitable position.

The Nurse.—Next, as to the nurse. It is advisable that the doctor should know the nurse, or at least be aware of her capabilities. Indeed, the medical man is often consulted on the matter, and asked to recommend a nurse. But all people are not alive to the danger of employing an incompetent nurse; very often engaging as nurse, without due inquiry into her capabilities, one who is recommended as a handy, motherly sort of person, and likely to give very little trouble in the house. Though these may be desirable qualities in a nurse, they are not the essentials, and every nurse should have had some training in lying-in cases. It is much more easy now than formerly to obtain properly trained nurses. It is good policy to obtain the services of the best nurse, for an insufficiently trained nurse is a potent factor for doing harm, and a good nurse a very valuable assistant during labour, especially in serious cases.

On arriving at the house, it is usual for the doctor to have a preliminary interview with the nurse. From her he may inquire when the labour set in, when the pains began, whether there has been any show, and how frequently the pains have been coming on; all of which a properly trained

nurse will readily answer. It is also important to know whether the patient slept during the previous night, whether the bowels have been properly relieved, either by medicine or by enemata (which a good nurse will look after, even without instructions from the doctor), and whether micturition has been natural and sufficiently frequent. Again, the nurse will very likely have been in the house for a few days previously, and will have seen to the preparation of the patient's breasts, by applying eau-de-Cologne or weak spirits and water to the nipples, and will see that they are not retracted.

The Bed.—During the early stages of labour the patient is usually up and about, and, generally speaking, she may be allowed to be about until the end of the first stage of labour, after which she should take to her bed. The nurse will also prepare the patient for an examination by the doctor, and get her in a proper position on the bed.

Now I must say a few words respecting the bed, for you should be in a position to instruct the nurse on the matter, if necessary. I have had this figure placed on the couch to demonstrate my points. Firstly, we will imagine that the patient is lying on her back in bed, and you require to make first an examination of the abdomen. The doctor stands on the right of the bed, the nurse on the opposite side. The bedclothes should be arranged in this way: First turn the sheet upwards over the patient's head, then turn the counterpane and blankets downwards, on a level with the lower part of the patient's abdomen, then turn the top of the sheet down, on a level with the upper part of the abdomen. This will enable you to examine the breasts. Having done that, to expose the abdomen you have merely the sheet to deal with, and by making a second turn downwards on a level with the first fold in the bedclothes, the abdomen may be fully exposed. If the examination is at all prolonged, it is well to place a shawl over the patient's chest and upper part of the abdomen. After your examination, replace the clothes by following the inverse order. This is a very convenient arrangement, and is far preferable to throwing the clothes down haphazard, as is so frequently done.

Next we will consider the arrangement of the bed when the patient is turned on her side during the later stages of labour. I will first deal with

the underclothes. As soon as the membranes rupture, you are likely to have a considerable mess in the bed from the escape of amniotic fluid. There are special sheets, a yard square, called sanitary sheets, consisting of absorbent cotton, wood-wool, or other absorbent material enveloped in gauze, intended for putting under the patient, at the side of the bed to catch the discharges. Where there is an excessive discharge of liquor amnii, however, these may not of themselves be sufficient. Among poor people, as in hospital out-patient practice, the usual thing is to fold an old sheet, blanket or counterpane, and put it under the patient. Otherwise, not only the bedclothes, but the bed itself may become saturated. It is a great advantage, and adds greatly to the comfort of the patient, if she be left high and dry at the end of the labour, let alone the damage done to the bedding, unless these precautions are taken. Beneath the pad it is usual to have a macintosh and drawsheet also on the bed. The under blanket and sheet are arranged precisely as in an ordinary bed, and over these is placed the macintosh, which should be large enough to go across the bed and slightly overlap at the sides, so that any excessive discharge is directed off the bed. In length it should be sufficient to reach from the shoulder-blades of the patient down to the knees. Above this macintosh should be placed a sheet, folded lengthwise so as to be a little longer from above down than the macintosh and entirely cover it. One end of this drawsheet should be brought over the right side of the bed, just sufficiently to tuck in. The overhanging portion on the opposite side of the bed should be tucked in. By this arrangement, as one part of this drawsheet becomes soiled, it can be drawn further to the right side of the bed, the soiled part rolled up and secured with safety pins, and a clean part exposed. Over the drawsheet is arranged the sanitary sheet described above.

Now, as to the arrangement of the upper bedclothes at the right side of the bed. During the second and third stages of labour, it is often necessary to examine the patient. To prevent the soiling of the upper sheet which covers the patient it is advisable to fix it with safety pins over the counterpane; and even to pin on a long diaper over it. By this means the clothes are kept out of the way. And to prevent undue exposure of the patient, napkins may be employed, which can be changed

as soon as they become soiled. There will then be no need to touch the bedclothes. In this way, at end of labour, the probabilities are that, if you are careful, you will have both sheets unsoiled, for all the discharges will probably be soaked up by the pad, and even if that be soaked through, the drawsheet and the macintosh prevent the fluid from going further and soiling the bed. After the first day, the macintosh can be removed altogether. To put in another drawsheet, proceed as follows:—having brought up the end of the soiled sheet close to the patient, to this pin the new sheet, with three or four safety pins, and merely draw on again, pulling the new sheet underneath. Unpin and remove the soiled sheet.

We will now go back to the time of labour. I have a few words to say concerning the "pulley." Formerly it was the fashion, and in many cases still is, to have a pulley fixed on the bed. As a rule, during a normal labour, the patient does not require to exert sufficient spontaneous effort to need a pulley, at least for the purpose of bringing her voluntary muscles into full play. But, provided the pulley be properly fixed, it may advantageously be called into use to assist the patient in maintaining a proper position in the bed. The best fixing point for the pulley is at the lower left-hand corner of the bedstead, not the head of the bed. The pulley may be a jack-towel, a Turkish bath towel, or a woollen scarf. By this arrangement, when the patient pulls she tends to bring the upper part of her body into the bed, and to keep the trunk across it. It is sometimes recommended to put a board at the bottom of the bed; but a hassock is better, the pressure on which, by the feet, tends to keep the patient in the proper position. Some patients roll about to such an extent as to render examination very difficult. If you find the patient is very apt to turn on her back (not a bad position—indeed it is the Continental and American posture), pass your left arm beneath her left thigh, take hold of her left arm, and bring it behind her back. This position should not be long maintained, as there is danger of cramp, but it is a very convenient posture for examination.

The Washstand.—I now pass to the arrangement of the washstand. There should be at least two basins, one for washing the hands, and another for antiseptic solution, which should always be kept

prepared in the room during labour. Some lubricant such as vaseline should be at hand. The lubricant may advantageously have some antiseptic combined with it. I have already dealt with the different antiseptics and the method of their use in a lecture which has been published.* I now simply content myself by drawing your attention to these tabloids of corrosive sublimate, some of which are coloured pink and some blue. They have been coloured pink at my suggestion, because a blue stain on the hands is more objectionable than pink, and it is necessary to have the solution coloured, that it may be readily distinguished from plain water. The tabloids are made of perchloride of mercury and pure chloride of sodium, and are of such strength that one of them to a pint of water gives 1 in a 1000 sublimate solution, the strength usually used for the hands. As a lubricant, perhaps, there is nothing better than vaseline, with which you may incorporate sublimate, by first dissolving the sublimate in glycerine, and then mixing the two together.

Napkins, &c.—Before I go any further, I must say a word or two about towels and such like. All napkins should be kept properly aired and warmed ready for use during labour. What I have further to add bears particularly on the poorer class of patients. A practice prevails in many country villages of providing a parish bag for poor patients, containing certain things which are required during labour—macintosh, napkins, babies' clothes, and so forth. It so happens that these bags are often sent to the cases which are doing worst, cases of puerperal fever, and so on. Unless great care is exercised in disinfecting, they are a constant source of danger. Therefore, always beware of the parish bag.

The Doctor's Coat.—The same remark applies to what I may call a "midwifery coat." Some doctors keep an old coat exclusively for midwifery cases, because, they say, it does not matter if it gets soiled. But it does matter a very great deal, because this dirty coat may be a means of carrying infection, and puerperal fever may dog the footsteps of such a doctor. I do not believe it is wise to keep a coat specially for midwifery cases. My advice to you is—never get your coat soiled, and, above all, never take a soiled coat to a midwifery

case. Every patient would not care for the doctor to remove his coat while attending her, though where any particular operation has to be done it is advisable. Of course you can, if you like, carry a separate coat made of washing material, and have it washed between each case. Apart from that, and especially in examinations in septic cases, it is well to guard your coat and shirt from soiling. The coat sleeve should have buttons at the wrist, so that the wristband of the coat can be turned back, the shirt cuff being similarly disposed of. To further guard the clothes from soiling, take a napkin, folded diagonally, hold one corner in the hand, and then roll it round the arm, and tuck in above and below; or put the napkin round the forearm squarely, tuck it in below, and pin it through to the coat above and below.

The Hands.—Next, be careful to thoroughly wash the hands and dry them. See that the nails are short and clean. Then soak the hands for half a minute or so in an antiseptic solution (sublimate 1 in 1000). Remember how very inadvisable it is to touch the patient with a cold hand. In cold weather it is not sufficient to merely hold the hands to the fire—they will not feel warm to the patient—but soak them for a short time in warm water. The same remark applies to the various utensils which are used, such as the bed-pan and the bed-bath. They should be warmed, either by pouring warm water into them just prior to use, or warming before the fire; then put a napkin or piece of flannel around the edge.

The Bowels and Bladder.—It is necessary to ensure relief of the bowels in cases where motions have not been passed freely. Early in the labour, a tablespoonful of castor-oil may be given in a little brandy and water, with a drop or two of spirits of chloroform to cover the taste. If the labour be too far advanced to allow the oil to act in time, a soap and water enema should be given. If the motions are very hard, it is better to pass 5 or 6 ounces of warm salad oil, and follow that up with a plain douche of water within a quarter of an hour. If the bladder has not been relieved and the patient cannot empty it voluntarily, you should always pass a catheter. When making the abdominal examination, you may notice a pyriform swelling at the lower part of the abdomen, indicating that the bladder is full. Either silk

* "Antiseptics in Midwifery." H. K. Lewis, reprinted from the CLINICAL JOURNAL.

elastic or soft rubber catheters are best. Be very careful to have the catheter aseptic, and as the water is passing, keep your hand on the bladder, and gradually exert pressure, so as to prevent any insuck of air. When the flow has ceased, put your finger on the end of the catheter and withdraw it. Hold the catheter vertically over the chamber with the eye downwards and remove the finger to empty it. Then wash the catheter by first running cold water through it in the same direction as the urine flowed, then steep it in an antiseptic solution, dry it and put it away.

Food.—Now as to the food to be given during labour. The food should be light, and mainly in the liquid form—such as beef tea, soup, milk, tea made with milk (preferably boiling milk poured on to the tea-leaves). The patient may also take biscuit or toast, egg beaten up in milk, custard, or milk pudding.

The Douche.—Next with regard to the douche. There was a time when douches were given very frequently, and for weeks after delivery, and there was a time before that when no one thought of giving a douche at all, even at the time of delivery. I think we now observe the happy medium, by giving the douche at the time of labour, and afterwards, not as a routine measure in ordinary practice, but occasionally if required. The object in view is to render the parts as aseptic as practicable at the beginning of labour, on the same principle as in making an abdominal incision for ovariectomy the skin is first washed and rendered aseptic. Especially is this precaution necessary in first labours, in which, be as careful as you will, you are likely to have lacerations about the vulva, which provide suitable sites for the absorption of septic material. In so doing you need have no fear of using a fairly strong antiseptic solution, e.g. 1 in 1000 sublimate, as there is practically no risk of absorption in giving the douche at the beginning of labour, before the rupture of the membranes. I show you here what is perhaps the best apparatus for the purpose. It is a hydrostatic tin, with hardened glass nozzle, which can readily be kept clean, and, being transparent, you can always see that it is so. The reservoir is furnished with a tap, and holds two quarts, so that for a 1 in 1000 solution four tabloids should be put in, and for other strengths in proportion. The temperature of the douche before labour should

be 100 to 110 deg. F.; afterwards, in order to cause the uterus to contract, it may be given at a higher temperature (115 to 117 deg. F.). Be careful to let some of the water run through the tube first, to warm it and to displace the air. In doing this it is necessary not merely to hold the tube downwards, but to raise the end, supervising the flow meanwhile by pinching the tube with your finger and thumb. A little vaseline should be smeared on the end of the tube before passing it into the vagina. Though the glass tube looks risky, I have never known one break except when dropped. These have been in use in the General Lying-in Hospital for many years without accident. Glass is preferable to the ordinary gum elastic nozzle, because, with the latter, if a clot is left in it, you cannot see it, nor is it so easily cleaned.

Vaginal Examinations.—We will suppose that you have seen to the points I have mentioned, and satisfied yourself that labour is progressing properly, and the patient has now arrived at the end of the first stage of labour. Then you have to consider what should next be done. It is often a question of waiting. I have already told you that you need not wait in the room during the early stage of labour. It is only when the membranes have ruptured, and when the head is coming down in the pelvis, that the presence of the doctor in the room is necessary. Make a thorough examination at the outset, but avoid too frequent vaginal examination, as it tends to irritate the patient, and does no good; as a rule a second examination need not be made until the head is coming through the pelvis. Especially avoid frequent examination after the douche has been given, particularly as each one is a possible means of conveying infection. Wash your hands and render them aseptic before each examination, and after this process avoid taking hold of articles of clothing or furniture, or putting your hands in your pockets.

Rupture of the Membranes.—Now we come to the point at which the membranes should have ruptured. Supposing you find the cervix is well dilated, you want sufficient resistance still left in it to cause the head to flex as it comes down. Therefore you had better rupture the membranes artificially, for which the best thing to use is a probe. Pass the probe along the finger, as in ordinary examination, and push the probe a *little* beyond the finger against the bulging membranes

during a pain; make sure first, however, that you are not trying to push it through the anterior wall of the vagina—in cases where the cervix itself is high up out of reach behind—by feeling the lumen of the cervix around the membranes; nor through the scalp of the child, where the membranes have already given way. Of course you will render the probe aseptic before use. This plan is better than notching the finger-nail, or using a hairpin, as is sometimes recommended.

Be careful to have ready a small basin, such as a sponge basin, to catch and scoop up the liquor amnii when its quantity is excessive. It is useful to carry in your bag, in case one is not to be had in the house, a vulcanite kidney-shaped tray, for into it you can receive the liquor amnii, as well as blood-clots as they come away after delivery of the child.

The Anæsthetic.—I next want to say a few words about chloroform. If I said that it is fashionable to administer it in labour cases nowadays, I do not think I should be far wrong. Most ladies ask that chloroform be given them, but the question is, when can it be administered advantageously? I answer, when the head is passing down through the pelvis and beginning to distend the perineum. It is not wise, as a rule, to give it in the early stage of labour. But chloroform is necessary, even in an early stage, when the pains are weak and almost continuous, where you find little or no progress is being made, and the patient is constantly crying out with pain. Chloroform given under such circumstances stops these continuous feeble pains, and in their stead the patient has strong pains at considerable intervals, which are much more effectual. In neurotic patients, chloroform is of decided advantage in the second stage of labour, but it need not be given to the stage of absolute insensibility, and should be withheld in the intervals of the pain. The patient, in fact, should be allowed to wake up between the pains, and you should renew the chloroform as the pain comes on again. It is very often advisable to give eau-de-Cologne with the chloroform, which enables one to give a minimum of chloroform while answering to the calls of the patient for a further supply. As the head is distending the vulva, the chloroform should be slightly increased, for the anæsthesia markedly assists the relaxation of the sphincter. But after the head has passed

the vulva, stop the chloroform altogether, or the patient may pass very deeply under its influence immediately the child is delivered. Where chloroform is given in considerable quantity or commenced in early stages of labour, there are risks attending it, for it leads to relaxation of the uterus subsequent to delivery. When chloroform is given be prepared to counteract hæmorrhage, especially where much chloroform has been administered. The same precaution should be taken after the administration of ether, which, on the whole, is safer when prolonged operative measures have to be undertaken. Have a hot douche ready, and give ergot, to contract the uterus, hypodermically if necessary. If a patient feels sick, ergot given by the mouth would not be absorbed, and would probably be rejected.

Support of the Perineum.—We must pause a moment, and go back to the time antecedent to the birth of the child's head, when the question of supporting the perineum arises. The necessity of affording support has been vigorously debated in days gone by, and there were many advocates *pro* and *con*. We have now, I think, reached the point when we know in what cases support of the perineum is useful. You must not exert such pressure on the perineum as to interfere with the due extension of the child's head, nor prevent the occiput from passing under the symphysis. It is, however, of advantage to fold a napkin and firmly press it over the perineum, but you must be careful that the pressure is not sufficient to prevent the head coming down.

There are certain rare cases in which the head is directed vigorously backwards under the influence of strong pains, and in which the perineum becomes much distended, while the head fails to be directed forwards through a rigid vulva in front of it. If in such a case sufficient pressure and support be not afforded to the back part of the perineum, there will be imminent risk of the head being forced out posteriorly, making a big rent in the soft parts. By judicious pressure, by lubrication, and in some cases by means of fomentations, you may obviate, in great degree, the rupture of the external parts.

In my next lecture I conclude my remarks on the care of the mother and child at the time of delivery, and shall then speak of the general management of lying-in cases.

LUNACY AND GENERAL PRACTICE.

SOME THOUGHTS ON THE DUTIES,
DIFFICULTIES, AND RESPONSIBILITIES OF THE
PRIVATE PRACTITIONER.

By T. SEYMOUR TUKE, M.B. Oxon.

GENTLEMEN,—When Dr. Keele asked me to read a paper to your Medical Society on a lunacy subject, I confess to feeling some diffidence, but he was kind enough at the same time to suggest what the subject should be, and I had great pleasure in trying to do what I could, as it is one which for some time has been a favourite one of mine, and on which I have thought much—the duties, difficulties and responsibilities of the private practitioner, when confronted with cases of insanity, and more especially developments of insanity in patients he is regularly attending, in their children and their relatives.

We, in London here, possibly do not feel quite so anxious on the subject as our brethren in the country, as special advice is always within reach, and we are able, as a rule, to avail ourselves of it, though there must still be many cases in which this is not practicable, and in which we have to rely on our own judgment, and take our own responsibility. But, gentlemen, my contention, and the principal aim of my paper is this, that the private practitioner should, as a matter of course, try and make himself conversant with the ordinary forms, signs and symptoms of insanity, to such an extent as to be able to cope with the cases that occur so often in our midst, and to be able more spontaneously and self-reliantly to give an opinion as to when it is necessary to seek special advice, or to act promptly, and without fear or favour on his own account, should circumstances compel him to do so.

It may seem a strong assertion to make, but I cannot help thinking that most of my hearers will admit that there is more than a modicum of truth in it, that there are few things that men in private practice dread more than being called in in a "lunacy case," or than seeing insanity developing in a patient under his care. He knows (especially after his first experience or two) the time it will take up, the anxiety it will cause him, the amount

of trouble, too often thankless trouble too, that he will have to go through, the worry and stress of knowing what to do for the best, and last, but not least I fear, too often he dreads what effect the Lunacy Act will have upon his action, and the present aspect of society, with regard to the disorder.

In the future I trust this may be entirely altered, and I repeat here what I said at Bristol last year, that I rejoice exceedingly that lunacy is to be made a special study in the examination of medical men: the fruit of it will appear in good time, not, I hope, only in the way of increasing the number of the existent single-patient private asylums, but in increased gain to society by the early and prompt recognition of the signs of trouble, by the prevention of insanity by good methods—hygienic and other—and increased knowledge of how to manage the young cases. I know well that insanity cannot be truly learned from books, lectures, or even by six months' asylum work. It needs constant daily intercourse with the insane for years (and even given that, one always feels there is so much more to learn); but the great advantage that the general practitioner has is that in many cases he has watched the patient from his childhood or his youth. I wish it were possible to add the study of insanity to the Board school curriculum. It would do more good than piano-forte playing. I feel sure that if not only all medical men, but all men, instead of regarding lunacy as something to know just a little about, something to know the name of, something to attend a course of lectures on, would only regard it (as Dr. Weatherly said), not as a crime, but as one of the commonest of all the scourges that afflict poor humanity, and one of the most absorbingly interesting of all disorders, from its very ungetatableness, that the world would be the better for it. It seems as though—while all has been progress around—the public comprehension of things appertaining to lunacy has stayed where it was, in ignorance, darkness, and chaos. It is not that those who are in charge of the insane have stood still or deteriorated, it is not that the places for their reception, both public and private, have not improved, but it is that the so-called public have not kept pace with the knowledge of the improvements, but prefer to stay fifty or sixty years behind in their ideas, and are loth to

leave off an ignorant outcry that is as foolish as it is undeserved. Were knowledge more general of this disease we should have fewer ill-assorted marriages; ill-developed, nervous children would not be pushed and overstrained; temperaments would be more carefully studied; life work would be more carefully chosen, and faddists would be relegated to a far less important position than they hold in many cases now. Men think and say that common sense can be their guide to a great extent, but if you have knowledge and experience to guide you, definite rules before your eyes, you will assuredly go less wrong than if you have common sense alone. For instance, it is terribly hard to decide when to take measures to restrain and protect from himself a man suffering from mental depression, and in how many cases this is left too late. I shall have more to say on this when I come to melancholia cases later on. Too often, alas! every one concerned seems to have lost what common sense he had, and the end is often disaster unspeakable. Lunacy confronts us everywhere, and I say distinctly and seriously that no man can be properly armed for the medical profession who has not some experience and knowledge of it. There are cases that help you and cases that do not; there are men who are glad to tell you all their troubles, and men who conceal them; men who show their suicidal, homicidal, perversions and uncontrolled propensities, and those who for long periods conceal them from every one; there are cases of volubility beyond all following, and those who do not open their lips, cases in which we can discover little or nothing, except from the friends, or from frequent observations. There are cases which the educated eye can see at a glance, cases that must be watched most carefully in their development. Cases where there are many ideas that are deranged, and those in which they are so plausibly concealed that it is most difficult to find anything to act upon; cases that can be easily discerned, and those that deceive, I had almost said, *καὶ τοὺς ἐκλεκτοὺς*. They have been classified, in a measure, from their generic resemblances, and so much help is given in recognizing them, but at the same time there are few cases exactly resembling each other, any more than there are men who are exactly alike. There are difficulties everywhere, and it is only by the most careful attention to everything that can help you,

backed by definite knowledge of your subject, that you can expect to surmount them.

Now take it that the case in hand is properly recognized, and your own mind made up about it. What comes next in the way of duties, and do the difficulties come in here too? Most assuredly they do, and those of the law are, I think, the least. There are friends to warn and manage, relatives (often in bitterest opposition to each other) to soothe and help, nurses to be found, provision for the patient's safety and that of those around him to be made, proper places to be chosen for your patient's bestowal, means of removal to be arranged—all apart from the procuring of the magistrate's order and the necessary papers. Of these I shall say but little. Some of us seem to be able to understand them at once, others seem helpless when called upon to use them. On this point I cannot do better than refer you to Dr. Blandford's excellent and lucid Lectures on Insanity, and to the special one on this particular subject.

There are, as we all know, two ways of dealing with private patients, as distinguished from paupers: one by urgency order, lasting seven days; the other, and this follows the last, by order made on petition, and accompanied by the certificates of two medical men. The urgency order is best signed by a relative, but can be signed by anyone over 21 years of age, provided a medical man can faithfully, in addition to his own facts, certify that it is for the welfare of A. B., or for the public safety, that the said A. B. should be under care and treatment, and gives adequate reasons. The same applies to the petition, and in both cases the signer of the document, when not related, should explain the circumstances under which he acts. The difficulty of obtaining "orders for reception" varies, I had almost said inversely, with the *savoir faire* of the magistrate. Some insist on seeing the patient, others form their judgment from the documents submitted to them, and weigh the evidence therein, not as though wishing to pick holes in it, but as though they believed it written down in good faith. Of course, much depends here upon the medical man, and on his powers of putting things clearly, and expressing his views and observations. One word about writing certificates: try and hit the happy mean between brevity and diffuseness; balance your

statements as carefully as possible; state clearly what delusions and insane acts are heard and seen, carefully explaining why you consider them delusions. Put your information derived from others as plainly as you can, verifying this as much as possible from your own observations, and making the one the balance of the other, weighing reasons, motives, and statements as carefully as possible before putting them on paper, and your certificate will do you credit, and assist you materially in the furtherance of your object.

I need say little, as you are probably equally well conversant with it, about the law on this head.

On the subject of paupers I would again refer you to Dr. Blandford. He says, p. 467:—

“The law enacts that the *medical officer of a poor law district*, on becoming aware of a lunatic, shall give notice thereof to the *relieving officer*, or, if there be not one, to the overseer. In the same way any person may give notice to the relieving officer and overseer. He is in turn to give notice to a justice of the county or borough, who within three days shall cause the lunatic to be brought before him, or shall visit him at his house, and shall examine him with the aid of a medical man.”

If the justice is satisfied, and the medical man certifies, the justice makes his order for his reception into an asylum for care and treatment. The pauper must either be in receipt of relief, or in such circumstances as to require relief for his proper cure. “A person who is visited by the medical officer of a union is to be deemed in receipt of relief. A pauper patient, therefore, is admitted into an asylum upon a justice’s order, one medical certificate, and a statement of particulars signed by the relieving officer.”

If the case is urgent, a constable, relieving officer, or overseer can remove the alleged lunatic to the workhouse of the Union, where the master can receive, relieve, and detain him, but the time must not be more than three days, and before the expiration of that time other proceedings may be taken.

Sometimes delay occurs in getting the patient to an asylum after the order, certificate, and statement have been signed. A certificate from the medical officer must be obtained to authorize this detention in the workhouse for more than fourteen days. After that time a fresh order must be obtained.

Wandering lunatics, paupers or not, may be apprehended, either themselves, or by a justice’s order, the lunatic to be brought before him. He calls in a medical man, and can order his conveyance to an asylum.

Neglected patients are also provided for; those, that is, of whom information is given on oath that they are either not under proper care and control, or cruelly treated and neglected. The justice has the same powers here, but he may also suspend the order for fourteen days, as in the last case, to allow the friends to arrange better for the patient.

Do not “forget, however, that it is lawful to restrain a lunatic who is dangerous to himself or others by virtue of the common law, apart from the lunacy statutes.”—BLANDFORD.

Time does not, I fear, allow of my going further into the Act, as regards property and persons, but I will say, in passing, that a new provision for caring for the property of the patient, apart from *Inquisitio de Lunatico*, is one of the useful things in that Act.

I propose, if time permits, to pass on now to a consideration of some of the special cases in which the opinion and the ready aid of the “usual medical attendant” (as the papers put it) are of the highest importance. First of all I put puerperal and lactational cases; these specially need careful watching, and well repay early attention and early recognition; while, on the other hand, few cases are more risky if they are let alone. Dangers to husband, child, and the mother herself may all arise with fearful suddenness, unless the medical man is wary about the early symptoms—the insomnia, the loss of appetite, the restlessness, and the alterations of affections and sentiments. Of course, I know that this particular disorder is probably best known about by the private practitioner, but care in the beginning is never so well repaid as in these cases. Competent and kindly nursing, separation and absolute quiet, diet and *régime*, all need attention, and our opinion may be asked as to positive removal from home and surroundings.

Next in order I place cases of melancholia, taking them as a group. These, alas! breed such fearful results, and are a fertile cause of the horrors which fill our daily papers. I do not hesitate to say that many of these crimes are preventible. How often we see in inquests the evidence that

much was known before the crime was committed, that with prompt action might have saved many lives, and many homes from misery and disgrace. Temporary insanity is all very well, but I say boldly that such a thing as temporary insanity exists most rarely, and that there is always, or nearly always, some premonition of the attack that the friends and the doctor should notice if they have the chance, and in many cases they have the chance. Cases in which there are ideas of imaginary diseases (especially syphilophobia), of impotence, of disgrace, bankruptcy, unpardonable wickedness, should not be allowed to go without supervision; and delay may cause endless mortification and infinite sorrow to all concerned. The liberty of the subject is one thing, but there are the liberties of other subjects dependent on this particular subject to be considered, too. Is not the safety and liberty of the poor victims of the suspected lunatic a thing worth thinking of? Are men and women to do as they like with themselves and their own without let or hindrance? Are we, because such a one does no harm, to let him marry, or if married, to leave defenceless women and children in his power, and imperil the lives and safety of many others besides himself? Act, if possible, promptly. Of course, I know it is hard indeed to know exactly when to act; but it is on the prompt decision not only of the specialist, but of the private practitioner, too, that so much depends in this matter, and no care can be too great, even at the risk of being thought too careful. It is never easy to say, "This man or this woman will attempt his or her life," but it is better to be too careful than to chance it, if we have a well-founded suspicion of it. Mistakes we may all make, all sorts of difficulties may confront us, relatives will not assist us, or may be divided in opinion. How often do we hear that medical advice was going to be obtained, that strangeness of demeanour was noticed, or even some absolute delusion known of, and then nothing is done, and the poor creature drifts to his death, dragging maybe several others with him, without a helping hand being stretched to save him and them.

A "fashionable" writer, speaking of a suicide in a recent pessimistic and in some ways most unnecessary novel, writes this:—

"The coroner's verdict always disposes of these cases comfortably in two words—temporary in-

sanity. All remorse, all despair, outraged honour, wasted love, together with the modern scientific theory of reasonable nothingness—life a nothing. God a nothing—when these drive the distracted human unit to make of himself also a nothing, temporary insanity covers up his plunge into the Infinite with an untruthful pleasantness." It is Satan who speaks, and they are cruel words; but, alas! to our shame I consider it is possible that they have been written, and cannot be said to be utterly false.

Cases of mania are, of course, more easy in a way; there is less doubt about them, but these are the resource of the practitioner as much as any in the way of management, nursing, and the like, and he may be called upon to treat them in their own homes. It is necessary to add that care should be taken not to confuse mania with the delirium of alcohol, that of typhoid and small-pox, and that precautions cannot be too carefully taken that harm to themselves and others is not caused by want of management and watchfulness.

Cases of food refusal, of insomnia, come into one's mind. These depend so much on treatment, knowledge of when to begin treatment, and especially how long to let the refusal go on without resorting to feeding, and when to recommend removal from surroundings, that it becomes necessary to call in special advice and experience to procure a good result.

Delusional, semi-sensible cases, and cases of the insanity of puberty, with or without masturbation. cases of the various "phobias," all require careful watching and supervision, for from the delusions so many things may arise that grievous harm may be done. There is no greater test of the proficiency of the practitioner than the tact with which he manages these cases, and the promptitude with which he decides on the course that is best to be pursued in them.

General paralysis, if unrecognized, may become the source of endless trouble and annoyance to friends, relatives, doctors, and all who come in contact with the patient. These do the most reckless things, sign papers, spend money, commit extravagances of all kinds, and do endless harm to themselves and others, in the stage of freedom and exaltation that is at last recognized as an early state of disease. Acquaintance with the signs and symptoms, with the various eccentricities, and the

restlessness and general strangeness of behaviour, is of the greatest service, and may be the prevention of much anxiety, difficulty and danger. Look for changes in habits, in affections, in sentiments, in manners, in punctuality, in business-like behaviour, in piety, and in the lesser signs of handwriting, power of doing small things and intricate work, and the physical signs, and take careful note of anything that can guide you, as outbursts of temper, irritability, incapacity to stick to one thing for any time, infirmity of purpose, and act promptly and at once when your suspicions are confirmed.

Time does not permit of more examples, though there are many things one would like to say about the insanities of puberty, that of heredity, senile dementia, and commencing decay, all of which are cases in which the family physician can by exercise of judgment and early recognition of the causes and effects do so much in the way of prevention, amelioration, and lessening of the troubles that ensue. I want to impress on the meeting that it is not to the specialist alone, not to the expert, to whom we have to look for the prevention and early recognition of insanity. It is on the "usual medical attendant," as the forms put it, the man who knows the family of the patient, that the first responsibility rests, and who ought to act fearlessly on the knowledge he possesses.

Gentlemen, these remarks may seem to you to be of little merit, and worthy of little attention. And you may think that I am only repeating what you knew before. But, gentlemen, we are passing through strange times, and stranger are to come. Unless the medical profession generally shows a brave and resolute front in this question of insanity, and shows that it knows something at least of the disorder, a state of things will arise that will end in chaotic confusion. Much has been written and said of late about insanity, and much abuse has been hurled at those who make it their special study, but it is chiefly started by those who either are ignorant of the subject, or wish to gain a certain notoriety or a certain amount of advertisement. *Humanum est errare*, mistakes must arise at times in all work that is human, but among the 92,067 lunatics under the cognisance of the Commissioners in Lunacy how many are there that could be said to be sane, and capable of managing themselves or their affairs; a great outcry is made over some

isolated case, and lo! all the whole body is blamed. The Government officials, whose one aim is equity, men whose lives have been devoted to the study of insanity, men whose private lives are of the most honourable, are held up to the populace as the vilest of mankind, while, on the other hand, suicides and crimes are condoned, and too late is the verdict given temporarily insane, and no blame attached to anyone.

It is to you gentlemen, to the family doctors and general practitioners, that we look. Teach your patients and their relatives the physiology of insanity, be ready yourselves with first aid, and it may be that instead of a lifelong regret, much satisfaction may be yours, in the sense of work well done, duties well performed, difficulties bravely and straightforwardly encountered, and responsibilities, heavy though they may be, unshirked and carried out in that good faith that the law allows to be your safeguard.

Read at the Islington Medical Society, November 26th, 1895.

A NOTE ON COCAINE ANÆSTHESIA

By J. JACKSON CLARKE, M.B., F.R.C.S.,

Assistant Surgeon at the North-West London Hospital, and
Pathologist at St. Mary's Hospital.

THE more one employs cocaine as an agent for producing local anæsthesia the wider the scope of the method appears to be. This ensues from the experience that solutions much weaker than those that were formerly employed are effective. The work of Schleich in Germany has of late given an impulse to the use of cocaine. This author has reminded us that even distilled water when injected into the skin at sufficient tension produces anæsthesia, but at the moment the pure water meets the nerve-endings, a smart of pain results. This undesirable effect is not observed if there is a small quantity of NaCl in the water. Schleich has utilized these facts and obtained anæsthesia by a solution containing only '02 per cent. of cocaine with a trace of morphia and a little NaCl. For my own part, I have not had constant success with any solution of less than $\frac{1}{2}$ per cent., but as

this is a much weaker solution than is used by many surgeons, I will venture to detail what seem to me to be the chief points in ensuring successful anæsthesia, and, at the same time, complete asepsis with the hypodermic use of cocaine.

The drug should not be kept in solution but in the solid form. Messrs. Allen & Hanburys have kindly made for me some tabloids, each of which contains 1 grain of hydrochlorate of cocaine and $\frac{1}{2}$ grain of common salt. These tabloids dissolve rapidly, and with ʒiij of water give a half per cent. solution, the whole of which may be injected with safety into an adult if the proper precautions are taken. In order to obtain a sterile solution, I use a porcelain capsule which is boiled with the instruments in the sterilizer. The capsule is emptied and a tabloid is placed in it, and then boiling water is poured on it up to the ʒiij mark. The capsule is then floated in cold water or placed on ice until the solution is cooled. Care is required to ensure an aseptic needle and syringe. The needle should be boiled with the other instruments. The syringe I have used for some hundreds of operations has the ordinary leather piston, which is certainly not ideal, but by keeping it solely for the one purpose and by washing it out with 1 in 20 carbolic before and after each operation, it can be kept aseptic. I keep the syringe in an air-tight metal case made for me by Mr. Hawksley, and in this way the piston is kept moist and always works perfectly. Perhaps a better syringe could be made on the plan of that used for mercurial injections in syphilis: the piston-rod is of glass with a piston of indiarubber drainage tube which can be easily cleansed with carbolic.

The mode of making the injection is also of importance. A case in which an operation involving the skin has to be done may be taken as an example. The needle is introduced very obliquely, so that the opening at the point remains in the corium. A few drops of the solution are injected and a slight wheal is produced, then after a few seconds the needle-point is pushed on for a short distance and a few more drops are injected. When in this way the corium is saturated around one part of the area to be incised it is withdrawn, and a fresh puncture is made at the opposite end of the anæsthetised area. By repeating the process of injection the whole circumference of skin around, say, a rodent ulcer may be anæsthetised.

The process is then repeated in the deeper tissues. In this way exactly fifteen minutes of anæsthesia are obtained. If the part is surrounded by the elastic tube the duration of the anæsthesia may be indefinitely prolonged. But after about fifteen minutes the constricting tube become irksome.

Old people are most likely to show symptoms of poisoning. In one case I observed it after solution containing $\frac{1}{10}$ grain of cocaine had been injected. The best preventive is to inject as above described so that not more than a few drops of the solution can enter a vein, to apply the elastic band until the chief incisions have been made, and to have the patient lying down. Brandy and amyl nitrite should be at hand in case of accident. In this way many minor operations, including stretching the anal sphincter and removing piles, can be performed, and in cases of emergency major operations may be carried out under cocaine alone.

REVIEW.

Transactions of the Dermatological Society of Great Britain and Ireland. (Lewis.)

The Dermatological Society was last year thrown open to the profession, and the volume before us is a report of its proceedings for the first year under the new conditions; also its rules and regulations, and a list of members. We must congratulate the Secretary, Dr. Stowers, on the excellence of the volume and on the splendid start that the really new Society has made (only those with experience know how much of such success is due to the energy of the Secretary). An interesting introduction by Dr. Pye-Smith (the first President); a valuable paper on thyroid treatment of skin diseases by Dr. Byrom Bramwell, and one on a comparison of the various methods of treating lupus vulgaris, by Dr. G. G. Stopford Taylor; a paper by Dr. Cagney on syringo-myelia and leprosy, form a good solid nucleus of most profitable reading, while the discussions of cases at the ordinary meetings fill the remainder of the volume, and are full of interesting and useful information to all interested in dermatology. We wish the Society and its proceedings every success which the importance and great prevalence of its subject-matter deserves. We think it would be well if a more uniform size (with other annual reports of societies) could be adopted in future years.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 15, 1896.

A LECTURE

ON

DISEASES OF THE TESTICLE.

Delivered at St. Thomas' Hospital, June 14th, 1895,

By SIR WILLIAM MAC CORMAC, M.A., D.Sc.,
F.R.C.S.,

Lecturer on Clinical Surgery to the Hospital.

GENTLEMEN,—In continuation of the subject of the last lecture, diseased conditions of the testis, I will now refer to innocent growths in the organ. I should like to show you these two cases of gummatous testicle, which you might examine later on. I have also several specimens from the museum of the commonest form of simple tumour of the testicle, namely, what is now called adenoma testis. This beautiful drawing as nearly as possible resembles one of these preparations: another water-colour drawing shows the uniform enlargement of the organ, with distinct encapsulation of the adenomatous tumour, and the thin layer of testicular tissue spread out over the surface. The tumour is completely within the distended albuginea, and the layer of tubular structure is spread over it. The disease appears to spring from the septum or rete testis, thence develops in all directions, and invades the entire organ. It is the common form of non-malignant tumour of the testicle. You will see from this specimen that it consists of very many cysts of variable size, some larger and some smaller, with a certain amount of interposed stroma consisting of connective tissue in various stages of development. The cysts are supposed to arise from an obstruction in the ducts of the organ. The stroma may be either spindle-celled or a combination of spindle and round cells, and there is also a considerable quantity of organized fibrous tissue in the midst of it. Cartilage, as you can see in the drawings and in the preparations on the table, is almost invariably found in these tumours, and in some myxo-

matous changes may take place in the cartilage and stroma. The cysts are lined with cubical epithelium, of which I draw you a rough illustration, and intracystic growths are often present; the contents are sometimes clear, thin fluid, sometimes blood-stained. When the tumour gets very large it causes thinning of the scrotal envelopes, bursts through its own capsule and then the superimposed structures, forming a fungating mass. At this period it shows a much more rapid development, and occasionally assumes a malignant type. Probably the embryonic element in the connective tissue stroma takes on an excessive growth, and thus transforms the tumour from a simple into a malignant growth.

These tumours generally occur in young adults, and are very frequently attributable to injury. As a rule there is neither pain or tenderness; they generally show steady, gradual increase in size. Sometimes the growth is more rapid, but usually the tumour has attained a very considerable size after a period of twelve months. It is firm and elastic, and in places presents evidence of fluctuation. The spermatic cord is not affected, or if it be, it is only invaded at a late stage. It does not appear to be involved in the growth. The function of the organ is lost, and if allowed to develop without check the tumour will become very large, apt to take on the characters of which I have already spoken, fungate through the scrotum and become malignant. These tumours are distinguished from hydrocele by their more globular shape and greater weight, and they are of course opaque. Their consistency differs at various points, in some places firm, at others fluctuating.

Another tumour which I need but briefly refer to is chondroma of the testicle, of which we have no example. A pure chondroma is exceedingly rare. It is of the hyaline variety. The rete is the part first affected. The tumour for a long time is limited by the albuginea and retains the shape of the testicle. There is no fluid in the tunica vaginalis; the cord is unaffected. Fibrous or calcareous transformation may occur. The tumour may remain, very slowly increasing, hard and painless, for an inde-

finite period, or suddenly take on active growth. As this tumour is prone to malignant change, the best treatment is timely castration. When of any size it is always found mixed with sarcomatous tissue.

There is also, in connection with the testicle, a very rare form of disease—fibrous tumour. We have no example of it here, and I have never seen one. It may spring from the albuginea or rete. The testis feels heavy, hard and nodulated, and in some cases the secreting structure is wholly replaced by fibrous tissue. It would be difficult to distinguish from a very chronic interstitial orchitis.

Dermoid cysts of congenital origin are occasionally met with. They usually arise in the body of the organ or may be on the surface. They are lined by true skin, and contain atheromatous material mixed with hair and foetal remains. It is doubtful how they originate. A swelling on one side of the scrotum will be noticed soon after birth. They may remain for an indefinite period without noticeable change; occasionally they suppurate, or even take on malignant action. Their congenital origin, and the evidence of the nature of their contents procured by exploratory incision, will determine their nature; and the treatment should be removal.

What is most commonly met with in hospital and general practice is a chronic enlargement of the testicle, due either to syphilis or tubercle. We have a number of preparations on the table illustrating both these conditions.

In referring to tubercle as it affects the testicle, I may recall to you that the French surgeon, Ricord, was one of the first to point out that tubercle, when affecting the genital tract, showed an extreme preference for developing in the epididymis; so much so, that he laid it down as a law that whenever tubercle was to be found in any part of the genito-urinary tract it was sure to show itself also, and to a greater degree, in the epididymis. This rule, although not absolutely correct, is practically true for the great majority of cases. In the young adult, during the period of active function of the gland, tuberculous disease is a common disorder, although it may be sometimes met with in adult life and in old persons. It is quite exceptional in this disease to find the body of the testicle affected without the epididymis, except in

those rather rare cases in children, in which tubercle in the miliary form is apt to occur, and in these miliary tubercle is to be found in the body of the organ, and the epididymis either not affected at all or only to a limited degree. But in the young person or adult the epididymis is the part of the genital apparatus which shows the largest amount of change, and in which the disease seems to originate. The body of the testicle becomes subsequently involved by extension of the malady from the closely adjoining epididymis. Out of some fifty cases you find both the epididymis and testicle involved in about thirty, though not, perhaps, to the same degree. In the remaining twenty the epididymis alone would be involved. Of course these figures are merely approximate, and serve only to give a general idea of the comparative frequency with which the epididymis alone is affected, and where the epididymis and testicle are both concerned. In twenty-two cases examined post-mortem, where the disease was probably further advanced, both epididymis and testis were found involved in ten, and the epididymis alone in twelve, a somewhat different proportion.

The disease is frequently bilateral. Examination of cases reveals the fact that it is bilateral about twice as often as it is single. When bilateral, it is usually much more advanced on one side than the other, and, as a matter of fact, the disease begins on one side and subsequently follows on the other. All parts of the epididymis may be involved. The disease does not confine itself to any one portion; the head, tail, or body between may be affected independently or together. The disease appears always to assume the caseous form. In the testicle the deposits frequently show a radiating character, corresponding to the fibrous framework of the organ.

The vas deferens is almost always implicated, and in a somewhat curious way. The portion between the external ring and the testicle is found to be the seat of considerable irregular thickening. In some cases this suggests a formation somewhat resembling a necklace, *i.e.* little knobs are felt in close succession, the deposit of tubercle taking place in the peri-tubular structure, and projecting into the lumen of the tube. The portion of the vas deferens near its termination at the seminal vesicle is also very commonly affected by the tubercle deposit,

while the intermediate portion is less liable to be involved.

With regard to the place where the tubercle is first deposited, the disease may commence in the seminal tubules and lymphatic spaces of the organ, or the deposit follows the course of the blood-vessels, more especially in young persons, in whom the glandular elements are not well developed.

One cannot say much as to the causation of the disease beyond speaking of the influences that tend to render individuals liable to tubercle in any part of the body, namely, any cause producing depressed vitality, as severe disease, prolonged illness, ill feeding, and neglect of the bodily strength, or excess of any kind. Any of these causes may so lower the vitality of the system that the bacillus finds a possibility of colonization and development. We also find the local damage done by injury attributed as a cause of the disease, or inflammatory changes occurring in the organ, because by these means the resisting power of the tissues is greatly diminished. But what is sufficiently remarkable and interesting is, that in a large proportion of cases of this disease, however developed, the affection seems to be of a local nature. Some thirty cases were recently carefully examined, and the possible presence of tubercle in any other part of the body carefully inquired into. Of these, sixteen had local tuberculosis combined with tubercle elsewhere, notably in the lungs, while in fourteen no evidence of tubercle in any other part of the body could be discovered. In a considerable number of post-mortem examinations, in which more certainty can be felt, the same proportion has been ascertained. In about half of these tubercle was found in the genital tract, especially the epididymis, but in no other part of the body. So that the law which, long ago, was laid down by the great French physician, Louis, that when tubercle is found in any part of the body, it is certain to exist in the lungs also, does not obtain in the case of the testis. Again, we find that only a small percentage of persons affected with pulmonary phthisis are subjects of tuberculous testicle. Phthisis in the lungs is exceedingly common, but only a comparatively small proportion of such patients are subject to tuberculous disease of the testicle.

The disease does not often occur before puberty, at least it is very rare before that time. As the result of examination of the bodies of a large number of

children, the testicles were found to be very rarely affected indeed. We may, therefore, say that in infancy and in young persons this disease does not occur in the organ, nor is it often found there in aged persons. The common period is at puberty, at the time of the commencing activity of the gland. Sometimes the disease sets in in rather an acute form, when one finds enlargement of the epididymis, swelling, pain and tenderness, with perhaps a little general fever. The disease runs a rapid course, and at the beginning resembles gonorrhoeal epididymitis in the acuteness of the symptoms—sudden severe pain radiating up inguinal canal, tenderness, the scrotum swells, becomes reddened, and fluid is effused into the tunica vaginalis—but these do not continue long. The acute manifestations subside in four or five days, and in three or four weeks you will notice the knobby, nodular condition in the epididymis, which is usual in tuberculous cases. In the acuter forms of the disease there is a marked tendency to early suppuration, the tubercular masses very soon break down and form abscesses, and the fistulæ follow which are so common in the disease.

This, however, is an unusual mode of origin. More frequently the affection is insidious, and does not declare itself by pain or irritation, sometimes not even by discomfort, and the patient often accidentally notices that there is something wrong.

There may be no apparent cause. The disease may follow on some slight injury, or a chronic inflammation of the urethra, which perhaps affords a suitable track for the bacillus to travel along. Any one of the general conditions to which I have already alluded may be causal, and the malady is frequently unnoticed in its initial stages. There is often some genital erethism, with erections and nocturnal emissions. There is some hydrocele fluid in about one-third of the cases. The degree to which the epididymis is involved varies much—it may be the globus major or minor, the body, or all three, while the testicle proper remains apparently normal. The masses of tubercle vary in size from a pea to a hazel-nut or almond. The disease is frequently discovered by accident, so little trouble does it occasion, and suppuration is sometimes delayed for a long period, even for two or three years.

After a time the changes I have mentioned as

taking place in the acuter form set in, in this instance more slowly; softening presently occurs in some of the tubercle masses; the skin over the part concerned becomes adherent. The caseous matter is discharged, and a fistula which continues to discharge a thin purulent secretion is the almost invariable result. The fistulous apertures are situated at the back and inferior portion of the scrotum, and they persist until the tuberculous matter is completely evacuated, when a cure may follow, or the disease extends to adjacent organs and becomes general.

I think you may assume that in some cases this disease is capable of cure. Cases occur in which there has been spontaneous evacuation of the tuberculous matter, or it has been scraped away, or it may become dispersed without suppuration; the conditions being otherwise favourable to recovery. The disease must, however, be treated in other cases by more radical means. When its local character is ascertained, the removal of the organ by castration will at the same time remove the disease and cure the patient, but unfortunately too many cases occur in which the disorder extends further up the seminal tract, along the vas deferens to the vesicula, involving perhaps by extension the prostate, engaging the coats and the mucous membrane of the bladder, and extending up the ureter to the kidney; thus it may become generalized throughout the whole genito-urinary apparatus. At the same time there may be tubercle elsewhere. Some are of opinion that the disease frequently finds its way down from the kidney to the parts below—to the bladder, the prostate, the vas deferens, and so on to the epididymis, but this is, I think, an unusual mode of transmission. The malady much more frequently spreads in an ascending direction.

The diagnosis in these cases is a matter of great importance, and with a sufficient amount of care should present no difficulty. You have to note the physical features of the individual, the age at which the disease is apt to arise, its slow, indolent progress, and the characters of the swollen epididymis and vas, which present an irregular outline, hard knobby projections being noticeable in some cases, while in others they are softer, and present signs of breaking down. When they have broken down you find the sinuses which result are always situated on the posterior inferior aspect of the scrotal wall,

contra-distinguished in that way from the softening down of gummatous masses which, when they break, present openings on the anterior surface of the scrotum. Further, you examine the condition of the prostate and vesiculæ through the rectum, and inquire as to the urinary function, to ascertain whether there be any evidence connected with it of disease of the kidney, or of alteration of function of the bladder, in increased frequency of micturition.

Now with regard to treatment. There is no doubt that when the disease is strictly limited to one testicle when, for example, there is no evidence whatever of tuberculous disease in adjacent parts, or in other parts of the body, as the lungs, the operation of castration, since the disease can be thoroughly eradicated, seems to afford a means of relieving the patient definitively of his trouble. In a large proportion of the cases the disease is at first purely local, and the operation affords a prospect that the patient may be wholly cured. It is important to bear in mind that where a sea voyage, open-air life, and prolonged careful treatment can be compassed, recovery without any operation may take place. Thorough removal of the tuberculous matter by careful scraping is often followed by satisfactory results. On the other hand, it may also well happen that although you have removed one testis the disease may have commenced, or be about to commence, in the other, and the removal of both is a very serious thing, which no patient likes to submit to. Still, cases do occur in which only one organ has been removed, and the patient has made a good recovery. Of course if the disease has affected other parts (such as the prostate, the bladder and the kidneys, or is present in the lungs) dealing with the epididymis is the least important element in the case and can have no effect upon the general condition of the patient. It is only in those cases where the disease is distinctly localized that the question of cure arises; when the disease has spread to the body of the testicle and to the vesiculæ, or the prostate, and the perineum perhaps is "riddled" (as it sometimes is by sinuses leading down to deeper structures which have become invaded), very little can be done. It has been recently proposed (and practised too in cases where one testicle is involved, and where the vesicula is implicated on the same side) that the disease

should be treated radically, by the removal, not only of the testicle and diseased epididymis, but by means of an incision in front of the anus, dissecting down to the fundus of the bladder and excising the vesicula as well. The operation has not, I think, been done in this country so far as I know, and I should imagine it is not likely frequently to be performed.

Apart from the local treatment, which may not only consist of castration of the organ but in the scraping out of the diseased parts, a method capable in some instances of doing a considerable amount of good, while in others, because of its inability to remove the entire diseased material, it is apt to be followed by either no advantage, or by a setting up an acute condition of the disorder in the parts around. In the less serious degrees of this malady, I have known the condition very greatly improved (in fact, as far as one can discover, cured) by such means as a prolonged sea voyage and very careful attention to general hygienic conditions and rules. I remember well one young fellow coming to see me whose testicle was the subject of a tuberculous deposit, the other testicle having been removed some time before for a similar state of things. This man had just succeeded to an estate of many thousands a year, and at my suggestion he lived at sea for some two or three years, and at the end of that time he became—so far as could be judged—quite well. So that these cases do sometimes recover, although on the other hand very many of them do not.

In arriving at a decision one may take into account that the obstructed tubules of the organ cannot transmit the seminal secretion, and the individual will be sterile when both testes are affected.

We now come to syphilitic affections of the testicle. It is not very long ago since all syphilitic affections of the testicle were confounded. Benjamin Bell was one of the first to distinguish between gonorrhœal epididymitis, for instance, and affections of the testicle proper which are due to syphilis. Before this time all venereal affections, both of the infective and non-infective character, and from gonorrhœa, were attributed to the same cause, notwithstanding that they have so little in common.

Syphilitic affections of the testicle proper are of

three kinds, the first in point of time being the congenital form. In congenital or hereditary syphilis the testicle does become affected with the disease. The frequency with which it occurs in children with syphilitic antecedents is a subject on which there is a difference of opinion. Some observers believe it to be met with as often as one of every three cases. I must say I have not observed it so frequently—myself, but it does occur with tolerable frequency. In such children, especially at about the fourth month from birth, you may find the testicles uniformly enlarged, the swelling being smooth, painless, and reaching perhaps the size of a pigeon's egg. It is not sensitive to pressure, and when cut into shows a dense, fibrous, whitish-yellow surface. It is a form of interstitial inflammation of the gland proper, and is apt to be followed by atrophic changes in the testicle. There cannot be much doubt, I think, where you meet with hard swollen testicles in a child in whom there are any other evidences of hereditary syphilis present, that the case is one in which the testicles have been involved by interstitial change, the result of the syphilitic poison. As a result the testicles often shrink and become atrophic, and there is a strong probability that in some of the cases of so-called undeveloped testes in children, who afterwards grow up in a sterile condition, the cause has not been a lack of growth in the testicle, but the shrinkage subsequent to syphilitic invasion.

The second form of the disease is that in which the affection of the gland appears in the secondary period of the disease. It then occurs generally in the form of epididymitis, which may be single, but is, perhaps, more commonly double. This variety of epididymitis generally appears about three or four months after the manifestation of the secondary eruption. On examining such a case one or more nodules of a hard, non-sensitive character are found, generally in the upper part of the epididymis. They are probably precocious syphilitic appearances in this situation, varying in size, and very indolent; their progress is insidious, and in many cases they pass altogether unobserved. There is no pain or tenderness connected with them, and very seldom any hydrocele fluid; in fact, little or nothing to attract the patient's attention. In some cases the symptoms are more acute, when, of course, attention will soon be directed to the

part. This acute condition lasts only a very short time, and the symptoms then become of the more usual chronic character.

In the condition commonly termed syphilitic orchitis we find the body of the testicle affected by gummatous deposits of the tertiary period of the syphilitic disease. Not only are these gummata deposited in the glandular substance of the testicle, but they may invade the albuginea, the tunica vaginalis, and scrotal envelopes of the testicle, as well as the spermatic cord, though not so frequently the latter. What seems to obtain, in the great majority of these cases, is that the epididymis is either not involved at all, or only to a much less extent than the testicle, and that the vas deferens escapes involvement in the vast majority of cases. A few instances are described in which there is uniform enlargement of the vas by interstitial deposit in its walls, causing thickening to two or three times its natural bulk; but in no case has anything been observed like the irregular nodular development in the vas deferens so commonly found in cases of tuberculous testicle.

Syphilis affects the testis in, perhaps, one in thirty or thirty-five cases; the statistics vary, but you may take these figures as approximately correct. As to the date in the history of the disease when it is most commonly met with, it is, perhaps, more frequent in the early or middle part of the tertiary stage, and is certainly most likely to occur in persons in whom the gland is in full activity of function. Kocher found that seventy per cent. of the cases were in men about thirty years old. It is also predisposed to by any previous injury or inflammation, and by the presence of tubercle.

As regards the history, we are told it is more likely to take place in severe cases of syphilis than in those of milder type, but this rule is by no means universal, for instances of syphilitic invasion of the testicle are met with where the patient has had a very mild attack of constitutional syphilis. Indeed, some have been observed in which the original disorder was passed by altogether unnoticed by the patient. Still we may take it that the disease is evidence in the main of a rather severe type of syphilis, and that it commonly manifests itself in the earlier period of the tertiary stage. I may here parenthetically remark that there is no pathological difference, no sharply defined line separating the periods of the secondary and tertiary stages

of syphilis; one glides imperceptibly into the other, and in the later secondary and earlier tertiary, the pathological changes which take place may differ somewhat in form but are really essentially the same in nature and character.

Let us now consider the symptoms of the disease. I have told you the time and period of its occurrence. Kocher states that in his experience seventy per cent. of the cases occur at about 30 years of age. The disorder generally first affects one testicle, subsequently its fellow becomes involved. It is essentially a very chronic, indolent form of inflammation—if such the invasion of the gland tissue by the gummatous deposit can be called. The testis becomes enlarged to the size of a hen's egg very often, or, as in this specimen, may reach the dimensions of a turkey's egg, but it very seldom exceeds this. The disease may present itself in the form of a general, uniform, fibrous alteration in the structure of the gland, involving the entire substance throughout, or as an isolated gummatous mass. In either case the swelling is very slow and gradual, except at the outset, when it is sometimes, but not always, accompanied by pain or tenderness. In the fully developed period of the disease the indolence (using the word etymologically) is markedly prominent. There is neither pain nor tenderness, and on squeezing the testicle there is no evidence of testicular sensation. In the earlier period, at all events, the scrotal tunics are quite uninvolved, and move freely over the swollen organ, which is capable of being readily moved about. On examining the epididymis you can sometimes distinguish it; but generally it is flattened out on the back of the swelling and cannot be made out as a distinct structure. In the large proportion of cases the epididymis is not involved at all, in others it may be to some extent involved by interstitial deposit between the tubules. On examining the vas deferens above the testicle, if you are able to discriminate it, you will find it in the great majority of cases absolutely normal; a fact proved, not merely by external examination, but by anatomical examination after removal. This, however, does not exclude general thickening or gummatous deposits which occasionally take place in the connective tissue of the cord, associated with alteration in the blood-vessels, from syphilitic changes in their walls. All this may occur, and yet the vas itself remain

intact. Contrary to what is so commonly found in tuberculous testicle, the prostate and vesiculæ as well as the vas are not engaged in the disease.

In, perhaps, one-half the cases of syphilitic testicle one will find fluid collected in the cavity of the tunica vaginalis. But although fluid may be—and frequently is—present in an early period of the disease, at a later date the two layers of the tunica vaginalis frequently become fused together. I here show you several specimens showing complete obliteration of the tunica vaginalis. In other cases there is an abundant hydrocele, but this condition is by no means universal. You have now got the general characters of this enlargement, its size, its gradual development, its sometimes perfectly smooth outline, either without any external protuberances, or “bosses,” or with such bossiness as may be very distinctly felt. This is due either to one or more of the gummatous nodules being deposited near the surface, or else—and perhaps more frequently—to the invasion of the tunica albuginea in an irregular fashion, with the cell proliferation which forms the essence of the disease. There is on the table a specimen which illustrates very well the invasion, not only of the testicle proper by the syphilitic changes, but of the albuginea, which can be seen as a distinct and thickened layer. The diffused, generalized orchitis is not pathologically different from the localized form, both are due to gummatous change; but in this specimen, for example, the disease shows a tendency towards fibrous development rather than to breaking down, and is in a very advanced stage of the complaint. Essentially the pathological conditions in both forms are dependent on the same cause. A point of great interest is the mode of distribution of these gummatous deposits—they may vary in size and number, may totally invade the gland, there may be only a single gumma or several gummata. They may also vary in size from that of a pin's head to a very large gummatous swelling. One feature of the distribution is that, as a rule, they are found in the anterior portion of the gland tissue, and what remains of the testicle is pushed backwards. In cases of tubercle the converse occurs, as also in most new growths; the gland tissue will be spread out in front of the tumour. This fact is of some importance in diagnosing these swellings.

As to the progress of the tumour, while it is

very easily controlled in the earlier stages by treatment, yet, if it go untreated, it either proceeds to general fibrification of the organ, as is illustrated in this specimen, or the gumma may break down and ulcerate through the scrotal wall, and thus evacuate itself, either leaving a fistula which will open on the anterior aspect of the scrotum or produce a fungus of the testicle. In either case the result will be the abolition of the function of the organ, and if both sides be involved it will result in sterility of the individual. Curiously enough, it has for a long time been contended that syphilitic testes never suppurate. Ricord, Gosselin, Virchow, were of this opinion, and in a true pathological sense they do not, of course, suppurate. The tissue undergoes a degenerative change and becomes liquid, and this fluid is capable of being absorbed under treatment exactly as the more solid gumma might be. Kocher laid it down that softening very rarely occurs, but these specimens on the table prove—if any proof were wanting—that the gumma does break down, and it then excites changes in the tissues around similar to those caused by an abscess in the same situation; the scrotal tunics become adherent to the surface, the skin becomes infiltrated and reddened, and finally ulcerates, forming an opening whose margins are thin, and undermined. There is irregular loss of substance in the interior; the skin projects over the margins of the opening, and its colour is bluish violet. The contents escape, leaving in some instances a “wash-leather like slough,” other cases ending in a certain amount of granulation material being formed. The deposit may form in the albuginea, and become protruded, constituting a so-called fungus testicle, or the fungus may spring from the gland substance itself. These changes are very much less acute than they would be in an ordinary abscess; scarcely any pain accompanies them, and hardly any constitutional disturbance. A softening gumma will prove amenable to treatment, even after these changes have taken place. On section, in a recent state, the gumma varies somewhat in colour according to its age; at the earlier parts of its existence it is of a more or less reddish-grey in colour, less firm and more succulent, showing slight patches of injection here and there. As it continues it becomes more dense and fibrous in character, dryer, harder, and

tougher. As a rule it does not show a definite border line between the healthy and the diseased parts; it is continuous with the adjacent structures, and we cannot enucleate it from its bed in any way. It will not break down under the finger-nail, as will a tuberculous deposit; it is not friable, it is dry, and there is no juice.

With regard to the microscopic characters: the cells are formed in the connective tissue; bundles of connective tissue are found developed between the tubules, the tubules being compressed. Their lumen is obliterated, they are separated from one another by the increase of fibrous material between them, and in the old standing cases they disappear entirely. The cells in the gumma which result from this pathological process are small and round, with little protoplasm and a large nucleus. In some cases there is not only a deposit of such cells in the stroma, but sometimes changes of a similar kind occur in the walls of the tubules themselves. Then of course in the testicles, as in other parts of the body affected with syphilis, there are marked changes in the larger blood-vessels; there is increased thickness in the middle and external coats from chronic arteritis; the internal is more rarely affected. Probably the infective material reaches the testicle by the blood-vessels. In the capillaries of the parts affected, a layer of fibrous adventitia is developed beneath the endothelium, presenting the appearance of a double layer of wall.

These changes when present—for they are not constant—differentiate syphilitic from simple inflammation. I have already said enough about the diagnosis of the hereditary form of the disease. In the secondary stage you may distinguish syphilitic epididymitis from either that of gonorrhœa or tubercle by its very different character, especially by its slow, indolent progress, and by the sharply-defined nodules which appear in the head of the epididymis as contrasted with the acute form of gonorrhœal epididymitis, and from the less defined character of tubercle, which is associated with evidence of tuberculous deposit elsewhere. You would try to verify your diagnosis by an examination of the rectum, and of the vesiculæ and prostate. Lastly, you have got a test for syphilis which is always useful, namely, the effect of treatment on the condition.

With regard to the differentiation of these tumours,

you will need to discriminate between syphilis, tuberculous disease, hæmatocele, and new growths in the organ, and this is not always an easy matter. In the case of tubercle there is the fact that the parts of the organ affected in this disease are different from those involved in syphilis; but a suppurating gumma might cause some difficulty when accompanied by a fistulous opening. You must therefore remember that in cases of syphilitic fistula the opening will be in front as well as sometimes behind, whereas in tubercle the openings are behind only, as a rule. In syphilis the body is always most affected and the vas is free. Again, in syphilis there is either no pain or tenderness or very little. In tubercle there is pain and tenderness, and you would find evidence of the disease in other parts of the body. The difficulty is greatly enhanced in differentiating hæmatocele. In hæmatocele the tumour varies much in bulk and consistence; the history, also, is often obscure. Still, the points I have mentioned will help you to a correct conclusion, and there is the possibility of getting a good deal of information either by a puncture or exploratory incision, as well as by the test of antisymphilitic treatment. To distinguish a syphilitic manifestation from new growth is often a task of still greater difficulty than in the last-mentioned group. A cancerous testicle may be recognized by its growth being more rapid than a syphilitic, and associated with more pain; emaciation may be a concomitant symptom. But in the early stage a diagnosis will be extremely difficult. A new growth would probably occur in one testicle only, while syphilis is most likely to affect both, though in a different degree. Further evidence may be obtained by trial of treatment, and the points I alluded to in a previous lecture on new growths of the testicle may be applicable here.

With reference to prognosis, the important circumstance is that if effective means be not taken to restore the organ by sufficiently early treatment, a patient with bilateral syphilitic orchitis will, in all probability, become sterile and subsequently impotent. But even in considerably advanced cases it is possible, by energetic antisymphilitic treatment, to restore the powers of the gland; to transform a hard, indolent swelling into a soft, normal testicle, and restore the fertilizing corpuscles to the gland secretion. The early forms of gummatous disease in this situation are

perhaps best treated either by local or internal administration of some form of mercurial preparation; while those in more advanced stages are best managed by doses of iodide of potassium. The quantity should be considerable—10 to 15 grains thrice daily, or even more, are generally needed. In a very advanced condition of the disease, after fibrous changes have occurred, you can imagine it is unlikely that you will be able to restore the function or healthy condition of the part. The final result will probably be an atrophic, sclerosed condition, while in some there may be a breaking down of the tissue, and formation of fistulous tracts. In such cases, when the organ is totally destroyed and the probability of any restoration of function by treatment has disappeared, the patient is best treated by ridding him of the source of his trouble by castration.

TWO CLINICAL LECTURES

ON

THE GENERAL MANAGEMENT OF LABOUR AND CHILD-BED.

LECTURE II.

Delivered at the Middlesex Hospital, November 8th, 1895.

By ROBERT BOXALL, M.D., M.R.C.P.,

Assistant Obstetric Physician and Lecturer on Practical
Midwifery to the Hospital.

GENTLEMEN,—At the conclusion of the last lecture we were considering the support of the perineum when the foetal head was extending. I now want to show you, by means of this model, what to do at this stage.

Delivery of the Child's Head.—The sheet is, you remember, at this stage pinned over the counterpane; the patient is lying across the bed. In order to prevent the patient from getting chilled, a napkin, which has been warmed, is placed over the lower part of her back, so as to hang down over the buttocks. The perineum is supported by another folded and warm napkin. One use of the napkin is to receive any fæces passed, as not

infrequently happens at this time. When this occurs, always take care to wipe the fæces away from the vulva, so as not to soil it, turning a clean part of the napkin for use each time it becomes soiled, and substitute a clean one as soon as the passage of fæces has ceased. Be careful always to have another clean napkin ready at hand, and as the child's head extends and passes the vulva, wipe its eyes, and clear the air passages from any accumulation of discharges on the face. Remember that with the next pain the trunk of the child will probably be born, and that if attention is not paid to the air passages, with the first respiration mucus and liquor amnii may be sucked into the air passages. Next, the finger must be passed around the neck of the foetus, to ascertain whether the cord is encircling it. If it is, you must promptly release it before the expulsion of the trunk. I have already told you how to do this. If the hand comes down with the head, you should generally keep it up. The chief danger of the elbow coming down with the head is that it may cause rupture of the perineum.

Delivery of the Trunk.—Occasionally you will find that the head and trunk are born with practically the same pain, and there is then very little chance of releasing the cord, should it be twisted round the neck. Fortunately in such cases it is generally a long cord, and, provided you prevent the child from being shot away from the mother there will be very little danger, for if you keep the insertion of the cord as near the mother as possible, there will be little risk of pulling on the placenta by means of the cord.

On the other hand, it sometimes happens that considerable delay occurs in the birth of the trunk. After the head is born the next pain or two does not bring it any lower down, and if unrelieved the child gets first blue, then pale. In such a case you are called upon to assist Nature. For this purpose take hold of the sides of the child's head. This can be done with one hand, if passed underneath the neck, and pressure be then made with the thumb on one side of the head and two fingers spread out on the other. If the anterior shoulder is hitched above the pubes, draw the trunk firmly backwards: as soon as this is released, the posterior shoulder will then come down over the perineum. If that fails, leave the uterus and, using both hands, catch hold of the child's

head on either side, release one shoulder and then the other.

Respiration of the Infant.—If the child does not breathe freely, rub it over with a napkin, pat its back, buttocks, and over the region of the heart. If this does not succeed, you must adopt some of the measures for establishing artificial respiration of which I have already spoken to you. See that the child gets air; extend its head by placing a folded napkin, or similar article, beneath it, or let it lie over the mother's thigh. The cord will be found passing out of the vulva, and if still twisted round the child's neck, loosen it at once, so as to permit the shoulders to pass through the loop, or pass the loop over the infant's head, taking care in doing this to keep the baby as near as possible to the mother. It is as well to have a napkin on the pad which has been placed for soaking up the liquor amnii, so that the child can lie on it. Then wait until the cord ceases pulsating.

Support of the Uterus.—Meanwhile, as soon as the child is born, place the left hand over the abdomen and supervise the uterus, in such a manner as to assist Nature. This is done by keeping the hand gently pressed on the abdomen above the fundus; be careful not to press merely backwards. I have seen many cases in which more harm than good has been done by faulty endeavours to "support the uterus," as it is called. To merely press the front wall against the back is not supporting the uterus. You need to support the fundus, which should be done by sinking the edge of the hand so as to hold the fundus in the palm, and pressing it in a line running at right angles to the plane of the brim of the pelvis, *i.e.* towards the anus. Continue to support the uterus with one hand, and feel the cord with the other, and stimulate the child, if necessary, so as to cause it to cry. When the cord has ceased to pulsate, the child should be separated. This can be done by the nurse, if she is efficient. If not, perhaps she had better support the uterus while you separate the child yourself. If she cannot do so properly, she had much better leave the uterus alone. To teach the nurse how to exercise proper support is not difficult. Taking up a position at the back of the doctor, while he is keeping his hand on the fundus, she should be directed to pass her hand, palm downwards, over the doctor's hand. If then

the doctor's hand is withdrawn from the fundus, the nurse's hand will take its place. She should be told to press gently downwards towards the anus.

Separation of the Child.—Now I will go back to the severing of the cord. It is usual to have a length of thread prepared, consisting of six to eight strands, knotted at either end, about 8 to 10 inches in length. Three ligatures are generally got ready; the material usually employed being unbleached thread, but tape or twine may be used. For my own part I prefer silk ligatures. The threads should be soaked in vaseline which has antiseptic incorporated in it. Or perhaps a still better plan is to soak the ligatures in the antiseptic solution which you use for washing your hands. The proper time to ligature the cord is when the child has been crying freely, and the cord has ceased pulsating. The ligature should be applied at least two inches from the navel, the reason being that a diverticulum of the bowel sometimes exists, so that in sloughing a fistulous opening might possibly be left. The proper way to tie the cord is by a surgical knot, in which the end, which passes underneath in one loop, becomes uppermost in the other, and *vice versa*. A knot which is liable to slip may lead to hæmorrhage. Slipping of the ligature is especially likely to occur when the cord is thick, and contains much jelly-like material. The first loop must be tied tightly, by pressing the thumbs together as you make the knot until the resistance is felt to give. It is well to cut off the ends of the ligatures as you proceed. Holding the cord in the clefts of the fingers of the left hand, and the ligature with the thumb and first finger, cut it in the palm of the hand, lest possibly a toe, or even the penis be amputated in the process. A second ligature is put on, not because it is necessary (except in some cases of twins), but because it prevents a mess in the bed from the blood squeezed out of the placenta. In cutting the cord be careful not to drag on the umbilicus, and keep the child's limbs out of the way. Hold it between the first and middle finger, and let it pass out between the ring finger and little finger, and again cut in the palm of the hand. The scissors, for choice, should be pointless (surgical ones). Having effected the separation, take a clean napkin and squeeze the end of the cord, to get rid of any blood which it may contain. If

there is no bleeding afterwards you may conclude that the vessels have been efficiently secured.

Removal of the Infant.—The child should now be handed to the nurse on a receiver—a piece of flannel for it to lie upon. To hold the child, put the palm of one hand beneath its shoulders, the fingers and thumb supporting the head; the other hand should support the buttocks. The nurse must see that it is kept warm, but that the face is freely exposed to the air.

Separation and Expulsion of the Afterbirth.—You must now again turn your attention to the mother. Put a warm, dry, clean napkin under her buttocks, to see if there is any hæmorrhage; keep her as dry as possible, not only from blood, but also from any liquor amnii which may have escaped with the delivery of the child. Then continue with your hand to supervise the fundus, and wait until the uterus begins to tighten up. Perhaps the first pain or two afterwards will not contract it sufficiently to cause complete separation of the placenta; but when you find a very firm contraction coming on, press your hand more firmly over the fundus, forcing it downwards and backwards, and in doing so squeeze the fundus and compress it at the same time. As it passes the vulva receive the placenta in your right hand. The membranes follow, though the amnion, as I have already explained to you, in some cases immediately follows the child outside, and remains folded around the funis. Sometimes the remaining membranes will come out without difficulty, while at others they still seem more or less adherent to the uterus, or may be nipped in the cervix. In that case, take the placenta as it comes in your right hand, and turn it round and round, so as to convert the membranes into a coil, which renders them much more likely to come away without tearing. And especially if any considerable traction be used will the membranes be likely to break off, the chorion often separating at the edge of the placenta. In such event you must pass your finger into the vagina or cervix to ascertain if there be another portion remaining in the uterus. In that case take hold of it with your fingers and twist it round, gradually pulling it away. The after-birth should be placed in a chamber half full of water. When the placenta is being born there is often a considerable amount of blood—6 or 8 ounces, sometimes more—and this blood should be put into a

separate utensil. Afterwards you can examine the placenta and the membranes to see that no portion is missing.

Administration of Ergot.—Immediately the placenta has been expelled the mother should be given a dose of ergot (1 drachm of the liquid extract); but, if the uterus does not contract properly and especially if the patient has had chloroform or ether, you may give 2 drachms at once. If, as often happens after an anæsthetic, the patient either rejects or is in such a state that she cannot absorb ergot given by the mouth, give ergotin or ergotinin hypodermically, thrusting the needle deeply into the muscles of the buttock. See that the ergot used is as fresh as possible; lapse of time often robs it of its strength. One of the best preparations you can use is liquor ergotæ ammoniata; for it keeps its properties better than the liquid extract of the British Pharmacopœia. Continue to support the uterus until it contracts down well, even after you have given the ergot. In first labours, unless very long and difficult, the uterus, as a rule, contracts very well indeed after the expulsion of the placenta; but in women who have had several children the case is different, and the uterus is much more likely to relax, and requires more support.

The Douche.—Wipe away any blood-clots that may be passed, then give the douche. It is better to give this with the patient lying on her back, with the bed-bath under her, than to have the patient on her side. While the douche is being given press on the uterus through the abdominal wall, though not so forcibly as before. Often during the process the uterus gets more or less pushed up, and sometimes it may be somewhat distended with the fluid. To my mind it is better to flush out the parts in alternate jets, expressing the fluid occasionally, while the stream runs continuously, than to give one squeeze at the end. At any rate, care should be taken that no accumulation is left.

Lacerations.—In drying the parts after the douche has been given, see if laceration has taken place about the vulva. If there have been any considerable tears, they should be sutured, and the best time to deal with them is at the time of labour. Remember that there may be very considerable laceration of the soft parts without showing externally; the perineum may have given

way without the skin over it being implicated. In order to ascertain this it is necessary to pass a finger inside the vulva. If there is a laceration of more than an inch in the oedematous condition of the parts, it is advisable to deal with it, or indeed with any tear which can be sutured. There may be tears not only in the perineum, but in the mucous membrane of the vagina towards the anterior part, which, you will remember, are all suitable surfaces for the absorption of septic matter, besides often causing much smarting and discomfort to the patient. Therefore, it is well to suture such rents at the time of delivery. For a tear in the perineum a needle in a handle is a great advantage, especially if the eye of the needle is made in the way I show you—the eye running through from one side to the other, instead of in the direction of the length of the needle; the needle should have a fairly sharp curve, the point being at right angles to the handle. Remember that the parts at the seat of the laceration are general considerably swollen, so that a large laceration one day may seem only a small one the next. This has a bearing on the passing of the ligature; you should not pass the suture too near the raw surface, otherwise it is very likely to cut through. Opinions vary as to the material which should be used for the ligatures. Silk is sometimes used, and when aseptic it is fairly good. Silver wire forms a very firm ligature, but has the disadvantage that the ends are apt to prick the patient and cause her considerable discomfort. To my mind, the best form of ligature is composed of silkworm gut, fairly thick, so that tension does not cause it to cut through; what is sold as sea-gut answers very well. The gut should be soaked in boiling water, to render it pliable. Still remembering that the parts are oedematous, you not only pass the ligatures deeply and well away from the laceration, but tie them tightly. Occasionally the laceration will extend into the rectum; and when the rectum requires stitching up it is better to do it with catgut, which becomes absorbed; silkworm gut requires removal.

Washing the Patient.—The douche having been completed, it is the nurse's duty to wash the patient. This she should do by uncovering first one part and then another. She must be particularly careful to remove all blood, etc., from the pubic hair, using for the purpose cotton wool

or linen, but not sponges, unless new or specially prepared.

The Pad.—Then the pad should be adjusted. Some people keep to the old-fashioned diapers, which are folded up, either lengthwise or diagonally, and placed between the patient's thighs, as I show you on this model. A substitute for diapers is now generally used, either in the form of absorbent cotton or wood-wool pads. The various forms of sanitary towel used for menstruation are rather small for this purpose. I show you here the pads in use at the General Lying-in Hospital, consisting of wood-wool encased in alembroth gauze to keep the wood-wool in place. It is a very comfortable and soft application for the patient, and is, moreover, inexpensive, the cost being less even than the expense of washing diapers. The pads should, of course, be burned after use. This reminds me that you should direct the nurse to remove all soiled articles out of the room as soon as possible. Soiled sheets and articles of clothing, such as night-dresses, if soiled, must be changed. The nurse, however, should see that the night-dress has been pinned up out of the way, and the lower parts enveloped in some readily moveable garment, such as a flannel petticoat tied by a tape round the waist.

The Binder.—Before the night-dress is put back to its proper position the binder should be applied. Many patients place great faith in binding, because they think it preserves the figure; but remember that preservation of the figure depends on the natural condition of the patient, and that damage to the figure occurs during pregnancy, rather than at the time of delivery or after. Indeed, it has been proved that the binder scarcely affects the figure at all; for in a certain number of persons the binder was used, and omitted in others under otherwise similar conditions; accurate measurements were then taken, with the result that scarcely any perceptible difference was found. This was done at the General Lying-in Hospital. Nevertheless the binder is an undoubted source of comfort to the patient, and for that reason should be used. I show you here a form of binder which is very efficacious and simple, and preferable to the complicated arrangements sometimes seen. It is a sheet of huckaback towelling, 1 yard wide and $1\frac{1}{4}$ yard long, doubled lengthwise, so that it forms a double sheet 18 inches in width. When

removing the clothes for the purpose of adjusting the binder, be careful to put a shawl over the patient's chest, especially if she be perspiring. The lower edge of the binder should be 4 inches below the great trochanter. Starting from the left flank the binder should pass over the abdomen, round the back, and again over the abdomen, ending on the right flank, where, after it has been tightened and all creases smoothed out, it should be secured by strong pins. The pins should for choice be straight ones, 2 inches in length, which are better than the ordinary safety-pin. If put in properly there is no likelihood of pricking the patient. Pass your finger under the lower edge of the binder, and pull it tight. The lowest pin should be inserted three inches below the top of the thigh-bone, and a second a similar distance below the top of the hip-bone, both fixing the binder tightly and prevent it from riding up. The third pin, being inserted on a level with the top of the fundus, should hold the binder still more firmly, and serve to support the uterus. No pad, pillow, or other such device is needed if the binder is adjusted in the way I have explained. A fourth pin, holding the binder loosely, is used merely to keep the upper edge straight, and if the waist is small a pleat may advantageously be made on either side with a safety-pin. Then the clothes may be put back into place.

The Washing and Dressing of the Infant.—

Until you have finished with the mother the nurse should not be allowed to attend to the baby, in order that she may be at the doctor's beck and call whenever required. Some nurses are anxious to get the washing of the baby over almost as soon as it is born. All that is necessary for the child while the mother is being attended to is to keep it out of the draught and properly covered up, but able to get air for purposes of respiration.

Now, the washing and dressing of the baby, though undertaken by the nurse, is a matter about which the doctor should at least know something. The temperature of the bath should be 96 deg. If the child be coated with much fatty matter it should be rubbed over first with vaseline, then the soap and water will remove all. Next as to the kind of soap. Several soaps are much vaunted as specially useful for infants, and mothers are often pestered with advertisements and samples of half a dozen special soaps. But many of these contain

an excess of alkali, and therefore are apt to prove irritating to the infant's delicate skin. Hard yellow soap is as good as, and preferable to, most fancy kinds. The eyes, nose, mouth and ears must be first attended to; they require tenderness in washing, and all creases and folds of the skin should receive special attention, and be thoroughly dried and well powdered. Oxide of zinc and powdered starch in equal proportions form a serviceable toilet powder, which is improved by the addition of a little powdered camphor—half a drachm to the ounce.

Then as to the care of the cord of the child. Having been carefully dried, it should be wrapped in a piece of absorbent cotton, kept in position by a length of flannel roller five inches in width and two feet long, which should be sewn at the back of the child, not pinned; any but safety-pins should be avoided in dressing a child. In private practice it is usual to wash the infant and re-dress the cord every day until it comes off; but sometimes the child is washed over but not actually put into the water until the cord has come away, which it usually does about the fifth or sixth day, though sometimes it remains on until the tenth. It is important for the nurse to know how to hold the child during the operation of washing—supporting the buttocks with her left hand, the back with the wrist, and the head with the forearm, the washing the baby may be accomplished with the other hand.

As to clothing, this varies very much in different households, but the simplest plan for a newly-born babe is to have all the clothes made opening at the back, so that by merely tacking them together, they can all be put on at once after the cord has been dressed; then a napkin folded diagonally is put on the buttocks, and over that what is known as the pilch, which covers over the legs as well. It is, however, very doubtful if there is any advantage in covering over the lower limbs in that way. I think it is better to let the clothes hang loosely. Immature and weakly children may with advantage be swathed in cotton wool. I have a caution to give with regard to napkins. To save themselves trouble, nurses are very apt to dry those which have been soiled, without washing, and re-apply them. This not only causes a urinous smell to pervade the room, but results in the child's buttocks soon becoming sore. Further, napkins

should never be washed in soda, unless they be subsequently well rinsed; and to ensure this it is often better to wash them out at home. A little vaseline or cold cream rubbed on the buttocks after they have been washed protects them to a certain extent from irritation of the aloine discharges.

The Doctor's Departure.—Before leaving the house, the doctor should always examine the child, to see that it is properly developed, that the cord is not bleeding, that the infant is not tongue-tied, that the limbs, feet and hands are perfect, and that there is no swelling about the scrotum. The mother will generally ask if the baby is all right, especially if some injudicious friends have been speaking to her during the pregnancy of possible deformities. Before leaving the house, the doctor should also assure himself that the uterus has properly contracted, that no unusual hæmorrhage is taking place, that the pulse has sunk below 100; and should give the necessary directions to the nurse as regards food and medicines, relief of the bladder, recording the temperature, exclusion of friends, ventilation of the room, suckling, etc.

I will now add a few words concerning the subsequent management both of mother and child.

Suckling.—First, a few words regarding the management of the breasts of the mother, a point which often gives rise to very considerable trouble. A child does not require to take the milk regularly from the very time it is born. It is sufficient for it to be put to the breast two or three times in the first twenty-four hours, to see if it can suck and to help to draw out the nipples. Until the milk comes in the breast, children very rarely require to be fed artificially. When the milk does come, the child should be put to each breast alternately, regularly every two hours, for ten minutes on each occasion. The nurse should see that the patient does not fall asleep with the child at the breast.

Artificial Feeding.—In cases where the mother is unable to suckle the child and it consequently has to be brought up by hand, the ordinary cow's milk in London is scarcely to be trusted. In the country, one part of milk may be mixed with two or three parts of water, with a little sugar of milk (which is better than cane sugar) and lime-water added. In London, humanized milk can now be obtained, and is as near an approach to mother's milk as can be procured. This does not require

diluting. It must, however, be given fresh, as well as at the proper temperature, namely, that of the human body. If condensed milk be employed it must be diluted with fifteen parts of water. When fed artificially, infants should have 4 oz. milk at each meal, and the bottle I show you is the best for the purpose—a boat bottle, with the teat on the end of it; a second teat should always be employed, and that not in use should be kept soaked in water. Bottles with long tubes are liable to act as syphons when once started, and the child has to take the milk, whether it wants to or not. Of course the bottle should be scalded between each meal. A convenient way is to use a feeding cup, with a teat on the end. Some weakly children are more successfully fed with a spoon.

Care of Breasts and Nipples.—Let us now turn again to the mother. After each application of the child to the breast, the nipple should be carefully washed and dried. Sometimes the breasts get hard and tender, from drooping at the sides. This can be rectified by supporting them with a folded napkin passing over the opposite shoulder and under the breast on either side. Hard breasts should be rubbed by the nurse with the hand, from the circumference of the breast towards the nipple. This rubs down the hard lumps and causes the milk to flow readily.

The Child's Breasts.—Occasionally the breasts of the child, even a male, become swollen and even secrete milk. In such cases, put a pad of cotton-wool on them and direct the nurse not to rub, touch or squeeze the breasts, or they will very likely suppurate.

The Child's Eyes.—Occasionally, also, the eyes become swollen, and if the case is not properly treated at the outset a severe attack of ophthalmia may supervene and the eyesight even may be lost. Having cleansed the eyes as the child was being born, you should, as an additional precaution, direct that a little warmed (1 in 2000) perchloride solution should be dropped into the eyes when the child is washed. But if in spite of this precaution you find that ophthalmia is pending, you must have the eyes frequently washed, first of all with warm water, and then drop a little antiseptic solution into them, subsequently smearing the eyelids with vaseline, to prevent them adhering to each other.

The Child's Buttocks.—When the child's motions

become green and irritable there may be considerable soreness of the buttocks, and you must direct the nurse to change the napkins frequently, and you must yourself correct the faulty condition of the intestines. Fuller's earth is a much used application for the irritated skin, but starch powder, with or without oxide of zinc, is perhaps better, and if to that is added a little camphor, as I have suggested, it is very efficacious.

Food.—The patient's food for the first day or so, until the bowels have been relieved, should be light and nutritious. If the bowels be not relieved by the second or third day, a mild aperient should be given, such as liquorice powder, or effervescing citrate of magnesia, or compound rhubarb pills, sometimes assisted by an enema. The bowels should be relieved before the middle of the third day. Fish, then meat, may be added to the dietary. Stimulants, as a rule, are not required while the patient is in bed, and, in fact, often cause febrile symptoms and discomfort.

The Lochia.—The lochia for the first three or four days are bright red and free, then become a pinkish, and later a brownish colour, and gradually diminish in amount. By the end of a fortnight, if not completely ceased, the discharge should be growing less and less and the colour disappearing. "Green-waters" are very seldom encountered where the case is treated in an anti-septic manner. A foetid odour at any time indicates danger and failure to maintain asepsis.

Relief of the Bladder.—The bladder must be kept properly relieved. When visiting the patient for the first day or two have the binder unpinned; see that the uterus is contracting properly, and that the bladder is not over-distended, for there may be considerable retention and yet a certain amount of water be passed. If the bladder be full, the uterus will be found carried upwards, and often lying considerably to one side or other, on account of the position in which the patient has been lying.

The Temperature.—The temperature of the patient should not rise above normal during the puerperal period generally, though often there is a slight rise when lactation commences, especially if the influx of milk is free and so sudden as to cause undue distension of the breast.

Movements of the Patient.—For the first day or two the mother should be kept lying low in the bed, and should not be propped up with pillows.

It is very necessary to enjoin quiet until the fifth day, and, especially if a patient be neurotic, it is advisable to keep her friends away. After the first ten days, or fortnight at latest, the patient thinks she should get up, but you should first ascertain that the lochia rubra have ceased, then make an examination. If you find that the uterus is going back to its proper position, and involution has reached the point it should, that the cervix has closed to such a degree that you are not able to pass your finger through it into the cavity of the uterus, you may let the patient get up tentatively, say to tea one afternoon. If the temperature then goes up, or if the loss becomes bright and free, the patient must be put to bed again. Should the mother have had febrile symptoms or lacerations, or be otherwise weakly, she should not be allowed to get up until after the lapse of a longer time. But in every case it is well to establish a practice of examining the patient before she gets up, because there may be some little mass retained, a portion of membrane or placenta which has been overlooked; or the uterus may have become retracted, and if you allow her to get up in that state she will experience gradually increasing discomfort and pain, and very likely hæmorrhage and leucorrhœa. Satisfy yourself that everything is satisfactory before the patient is allowed to get up, otherwise she may have to return to bed for a much more protracted period.

SUGGESTIONS FOR A SERUM TREATMENT OF SMALL-POX.

SMALL-POX, thanks to vaccination and re-vaccination, has been robbed of much of its virulence as an epidemic disease, but it still rages in truly epidemic form, attacking most severely the unvaccinated individuals of the community and those who have neglected re-vaccination.

Judging by analogy with other diseases, we may, I think, take it for granted that vaccination in man produces anti-toxin in his blood, which renders his system refractory to the infection of small-pox.

We may also take it for granted that in the calf after vaccination and the production of cow-pox, the animal's blood contains an anti-toxin

making it refractory to cow-pox, which is the same disease in the calf as small-pox is in man.

I would suggest that this protecting anti-toxin in the blood of the calf lately recovered from cow-pox, should be used in the same way as the serum of the horse, rendered immune by repeated small doses of the toxin of diphtheria.

A certain dose of serum containing the anti-toxin of diphtheria cures diphtheria in a number of cases; why should not the serum of the calf lately recovered from cow-pox contain an anti-toxin which will cure small-pox?

An experiment of this sort should be only undertaken by experts; it is likely to do no harm should it fail, and for my own part I think that a large number of lives of small-pox patients could be saved by the injection of calf-serum from an animal immunized to cow-pox by vaccination, and which has lately recovered from that disease.

These injections should take place as early as possible in the disease, so that the anti-toxin should have the chance of doing its work before the microbes of small-pox have developed a large quantity of small-pox toxin.

Cannes.

ALFRED CARR.

THERAPEUTICAL NOTES.

Bronchitis, Herpetic.—According to Lance-reaux there is but one remedy which will calm the cough of herpetic individuals—a cough occurring spasmodically and preceded by a tickling sensation in the throat. This remedy is sulphate of quinine, in doses of $23\frac{1}{4}$ to 31 grains for adults, and 6 to 12 grains for children, taken in two or three doses half an hour apart. To obtain a satisfactory result it is essential that the patient feel buzzing in the ears, vertigo, headache, etc. This result is ordinarily apparent in three or four days, but the treatment must be continued during the same space of time in order to prevent the return of the cough.

Gazette des Hôpitaux.

Dry Seborrhœa of the Scalp.—R. Betanaphthol, $4\frac{1}{2}$ grains; camphor, resorcin, each 3 grains; sulphur precipitate, 46 grains; pure vase-

lin, 5 drachms. Use as an ointment twice a week, removing any excess with a handkerchief. Next morning clean the head with a little petroleum ether, or, if too irritating, use the following:—Liquid ammonia, $2\frac{1}{2}$ fluid drachms; rum, $1\frac{1}{4}$ fluid ounces; tincture of quinine, tincture of rosemary, each one fluid ounce; walnut-leaf infusion, $6\frac{1}{2}$ fluid ounces. If the seborrhœa is very oily, apply the following lotion three times a week:—Sulphur, $6\frac{1}{2}$ drachms; camphorated alcohol, $1\frac{1}{2}$ fluid ounces; pure glycerine, $1\frac{1}{4}$ fluid drachms; distilled water, $6\frac{1}{2}$ fluid ounces.

BROCO, *Jour. de Méd. et de Chir. prat.*

REVIEW.

Treatment of Pulmonary Consumption. By Drs. HARRIS and BEALE. (Lewis' Practical Series: Price 10s. 6d.)

We have read this book with more than ordinary care before criticising it, because of the immense amount of divergence of opinion on its subject, and also because of the pernicious influence which climatic treatment indiscriminately applied has exerted. After this careful perusal, we are very pleased to be able to recommend the work very highly. The pathology of the disease is more clearly and succinctly given than in any other book with which we are acquainted, and upon this solid foundation is built a thoroughly satisfactory superstructure of well-considered methods of treatment. The main idea underlying the work is that the patient must be more studied than the bacillus, and consequently the suggestions for treatment are practical and common sense. Bactericidal remedies, so often recommended, are placed in their proper place—never satisfactory alone, but occasionally helpful to other remedies. No one who turns to the book for help will go away disappointed, every chapter nearly teems with useful hints and prescriptions for all sorts and conditions of patients. The book fully sustains the reputation of the series as "practical," and we strongly recommend it to our readers.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 22, 1896.

CLINICAL LECTURE

ON

CHRONIC LEAD POISONING.

Delivered at University College Hospital, January 9th, 1896,

BY

G. V. POORE, M.D., F.R.C.P.,

Physician to University College Hospital; Professor of
Clinical Medicine and Medical Jurisprudence,
University College, London.

GENTLEMEN,—In Ward 8 you have frequently seen a patient who was admitted to the hospital on September 26th, 1895, suffering from obstinate constipation and great muscular weakness. He is a painter *æt.* 34, and stated on admission that his bowels had not acted for ten days, and he informed us that on three previous occasions (in 1879, 1888, and 1891) he has suffered from obstinate attacks of "colic" and constipation. As he is a painter by trade, and has a well-marked blue line upon the gums, we have little hesitation in attributing his troubles to lead poisoning. He thinks the present attack was determined by his working, during very hot weather, at painting the vaulted ceiling of a room, all the windows of which were closed.

The attack of colic in September was accompanied by other symptoms from which he has not previously suffered. He stated that for three weeks prior to his admission he had been feeling weak and ill, and three days before his admission he found that, owing to weakness in his arms, he could not lift a cup to his lips.

On admission his complexion was sallow and muddy, and he was much emaciated. He could not extend the wrist nor, owing to weakness of the deltoid, could he raise his arm. He could not as he lay in bed change from the recumbent to the sitting position without assistance; he straightened his back with difficulty, he could not stand without help, and was quite unable to walk.

VOL. VII. No. 13.

In short, every muscle of the limbs and trunk was in an extreme degree of paresis. All the muscles were soft and flabby, and to a certain degree wasted. I use the term paresis instead of paralysis, because every muscle seemed to be capable of a certain degree of feeble movement, and all of them responded to the faradic current. There was no extreme wasting of any muscles, except perhaps the erector spinæ on the right side. The face rather lacked expression, but no definite paralysis of face or lips or ocular muscles could be detected.

He feels when he is touched, and can tell when he is pricked with a pin, but nevertheless I doubt whether his sense of pain is very acute, because when the hair of the leg was suddenly pulled he did not flinch to the degree which is usual in a normal subject.

All the nerve trunks in the upper limbs are exceedingly tender, and it is evident that the nerve trunks are much more tender than the muscular masses.

When I first saw this patient I was much struck by his voice, which was feeble and toneless, without being hoarse or stridulous. The voice was of a character to excite a suspicion of laryngeal paralysis, and on examination with the laryngoscope we found well-marked immobility of the left vocal cord, which was not adducted to the middle line during phonation.

The urine since his admission has been of average quantity but pale, of low specific gravity (fluctuating between 1.014 and 1.008), and up to November 5th contained a trace of albumen, which has not been detected since that date. The urine, kindly examined by Dr. Kenwood on January 9th, gave evidences of a slight trace of lead only.

The temperature has been normal except for a few days, when he suffered from slight tonsillitis.

The hæmocytometer on October 1st showed that the red corpuscles were about eighty per cent. of the normal.

For the first week of his admission he suffered from constipation and abdominal pain, but since

that time the alimentary tract has afforded no evidence of disease.

In some respects this is not quite a typical case of lead poisoning, although there are no symptoms which have not been caused by lead again and again.

As to the paralysis from lead, you are most of you probably aware that the typical form of lead paralysis is "wrist drop," in which the extensors of the fingers and wrists are paralysed on both sides. As a rule the paralysis from lead is limited to the forearms, and to the muscles named. The supinator longus and the intrinsic muscles of the hand usually escape. The paralysis is generally symmetrical, but while both limbs are implicated, it is usual to find that the right arm is affected earlier, and rather more severely, than the left. The affected muscles waste rather rapidly, and in a severe case of long standing give, when tested with electricity, the reaction of degeneration.

With regard to this "Reaction of Degeneration," please remember that the important fact to bear in mind is that the muscles do *not* respond, as normal muscles do, to the faradising current, while they respond with undue readiness to the continuous galvanic current.

The non-reaction of the muscle to faradism is said to be due to the degeneration of the intramuscular nerves. This explanation is probably correct, but it is well to remember that a paralysed muscle which is recovering will sometimes give the reaction of degeneration at a time when it is responding to the stimulus of the will, *i.e.*, it is the seat of voluntary contraction when artificial contraction cannot be evoked by faradisation. This may be due to the fact that the intramuscular nerves have regenerated on the under-side of the muscle, where the faradic stimulus is not easily applied. Why these muscles should show an *increased* irritability to the galvanic current it is difficult to say, and still more difficult to say why the reaction to the galvanic current shows a *qualitative* as well as a *quantitative* change. This qualitative change consists in the fact that the muscle reacts to anodal opening or anodal closure before it responds to cathodal closure, which is the first reaction to be obtained in health. These qualitative changes are interesting, but their importance from the clinical point of view is as yet not understood, and I want you to cling to the

main fact of these reactions of degeneration, which is that *the muscle which does not respond to faradism, responds readily (sometimes too readily) to galvanism*. The degeneration of the intramuscular nerves is brought about by extinction of the motor cells in the anterior cornua, so that no stimulus can reach the muscle from the brain, or by a block in the nerve itself due to neuritis, morbid growth, extravasation of blood from bruising, or the actual division of the nerve. So soon as a motor nerve is severed or blocked by the processes named, degeneration begins to extend centrifugally until it reaches the muscle. In mixed nerves which serve both sensory and motor functions, a blockage of any kind occurring in the trunk probably produces a certain amount of centripetal degeneration, as well as centrifugal; but this latter is far more difficult to demonstrate.

The patient whose case we are considering is not to be regarded as a typical example of lead paralysis. In him the paralysis is of very wide extent, but it resembles other cases of lead paralysis in this, that it began in the forearms, and that the deltoid muscles are among those which are most profoundly affected. Scarcely a muscle on his body has escaped; all are feeble in an extreme degree, but none appear to be totally paralysed, and all of them, which are accessible, respond to a *strong* faradic current.

The paralysis of one vocal cord is to be regarded as exceptional. Cases of such paralysis from lead are recorded, but I do not recollect having personally observed it before. Naunyn states that in horses which work in lead mills paralysis of the abductors of the vocal cords is common, and that, not unfrequently, this necessitates the performance of tracheotomy. Paralysis of the left vocal cord in an adult is a physical sign which raises a strong suspicion of aortic aneurysm; but repeated examinations of our patient to this end have always given negative results.

How profound is the change produced in the human body by the continued introduction into it of small quantities of lead! Lead holds an important position in the Pharmacopœia on account of its styptic qualities, and is frequently administered in cases of internal hæmorrhage. It has a reputation also as a valuable astringent when applied externally, and the solutions of the

salts of lead are probably antiseptic to a considerable degree. This poisonous metal is retained in the Pharmacopœia for its styptic and astringent qualities—qualities which are due to its stimulating action upon the muscular coats of the small arteries and arterioles. It is said to have a similar action upon the muscular coats of the intestines, and to cause in this way the phenomena of "painter's colic." Our patient on admission had been constipated for ten days. His intestines, and probably his arterioles, had been the subject of a muscular spasm so sluggish and inert as to be regarded as permanent. When the full styptic effect of lead has been produced the patient is in great danger if its administration is continued. It seems probable that when lead produces a toxic effect the calibre of every arteriole in the body is diminished. If this be the case, can we wonder that the nutrition changes are profound? Absorption from the alimentary tract is diminished, the eliminations of waste products by the kidney is seriously checked, and the starved tissues tend to degenerate, so that in many places we find that the connective tissues tend to increase at the expense of gland cells and nerve tissue.

Dr. Thomas Oliver, of Newcastle, in his Goulstonian Lectures on Lead Poisoning, gives an excellent series of drawings of the histological changes in lead poisoning. In the kidneys the shedding of the epithelium of the tubules is accompanied by interstitial overgrowth of connective tissue; in the liver cirrhotic changes occur; the fibres of the nerve trunks are pressed upon by fibroid overgrowth; and similar changes also occur in all parts of the body. The kidneys are very liable to be affected in lead poisoning. However the poison be introduced, whether by the stomach, by inhalation, or by the skin, the kidneys must perform an important part in eliminating it, and the contraction of the renal vessels seriously damages the organ, and thus its physiological action of eliminating the end products of metabolism is damaged, and we are liable to get gouty symptoms in those who are otherwise predisposed to gout, and not unfrequently uræmic symptoms, inclusive of epileptiform convulsions. It is evident that when the kidneys are damaged, the danger caused by the poison circulating in the blood is enormously increased. It is also evident that if a worker in lead acquires a granular interstitial

nephritis, the danger he runs from his occupation is increased, and his liability to suffer from recurrent attacks of lead poisoning is very great. The really operative dose of a poison is the balance between the amount absorbed and the amount eliminated. If elimination keeps pace with absorption no symptoms are caused, but if elimination be slower than absorption, or if elimination be checked by disease of the kidneys, then, as we all know, very small doses of such poisons, as mercury and opium, are liable to produce most untoward results.

The physiological action of lead salts being to cause contraction of the vessels and intestines, it almost necessarily follows that so soon as absorption takes place elimination is checked, and in this fact we find the explanation of the insidious nature of lead poisoning. There seems to be no quantity of a lead salt too small to produce symptoms, *provided it is taken continuously*.

In the patient whose case we are considering there is no history of any attacks of gout, but it is well known that workers in lead are very liable to gout, and that sufferers from gout are peculiarly intolerant of lead. It was Sir Alfred Garrod who first demonstrated in this hospital that the administration of a few grains of lead acetate to a gouty patient almost invariably produced a paroxysm.

This liability of the lead worker to suffer from gout has not been observed in Edinburgh to the same extent as in London, and lately Dr. T. Oliver has shown that the lead workers of Newcastle, and the northern towns generally, enjoy a similar comparative immunity from gout. It used to be thought that the difference in the liability to gout in North and South was to be accounted for by the fact that while the northerners stimulated themselves with non-gouty whiskey the southerners used beer, the reputation of which as a gout-producer is considerable. This difference in habits may partly account for the difference in pathological tendencies, but that it is not the sole explanation seems certain from the work of Dr. T. Oliver and others. The true explanation of this strange difference is still to be sought.

The symptoms produced by lead are protean, and there is not a single symptom which is constant unless it be the sallow pallid complexion. The anæmia produced by lead is extreme, and

the presence in the blood of a very small quantity seems to cause a rapid diminution of both hæmoglobin and red corpuscles.

The lead, after absorption, circulates with the blood, and its effect seems to fall upon different organs in different individuals. We all have our vulnerable points, and these are not the same in any two persons. There seems nothing unreasonable in supposing that any part of the body which has been damaged, and the nutrition of which has been impaired by previous disease or injury, is likely to constitute a vulnerable point. We know, also, that persons have a family tendency towards chest diseases, vascular degenerations, insanity, etc. Did time permit one could bring forward many familiar examples of these propositions. Occasionally it may happen that the parts most used are the ones to suffer most, and this explanation has been offered of the tendency of painters to suffer from wrist-drop. The tendency of different individuals to be differently affected by lead caused Tanquerel des Planches to classify his cases, and to speak of (1) lead colic, (2) lead paralysis, (3) lead arthralgia, and (4) lead encephalopathy. Lead arthralgia, which is not very common, would probably resolve itself nowadays into gouty pains and pains due to spinal sclerosis and neuritis. Lead encephalopathy (headache, convulsions, tremor, optic neuritis, and atrophy) may be primary, but is more probably, in most cases, secondary to chronic kidney trouble.

What we have to remember is the great fact that the symptoms of chronic lead poisoning are varied and far from uniform, and, in short, that lead may be the unsuspected cause of chronic troubles affecting nutrition generally, or the digestive, renal, or nervous organs.

Many poisons manifest a peculiar affinity for the cells of certain tissues, which stand in relation to the poison in a position analogous to that of preference shareholders in a trading company. Few, if any, poisons, however, have an *exclusive* affinity for the cells of any tissue, and it is usual to find that after those cells for which the poison has a "preference" have been supplied other cells in the body begin to get some of the dividend. Lead is more impartial in the bestowal of its injuries than most poisons, and although "lead colic" is the commonest symptom we have seen

how insidious is the action of lead, and how it may affect every organ in the body. It has been said that "lead has a peculiar affinity for nerve tissue, but analyses made on the bodies of animals which have succumbed to lead poisoning show that it is universally distributed, and that every part of the body can be made to yield lead." The distribution of the lead in the body varies considerably.

As lead poisoning is insidious in its onset and very protean in its manifestations, it becomes important to consider what symptoms, if any, are to be regarded as pathognomonic. The blue line which appears on the margin of the gums next the teeth, often spoken of as the lead-line (and which is due to the precipitation of lead sulphide in the tissues of the gums, by the action of the hydrogen sulphide formed by the decomposition of albuminous food particles in the mouth), is the most valuable aid to the certain diagnosis of lead poisoning. Other things being equal, the patient who generates most hydrogen sulphide in his mouth will have the best marked lead-line; while those whose mouths are inapt, either by design or accident, for this decomposition, may suffer from lead poisoning without any lead-line upon the gums. Persons who have no teeth, or those who are blessed with very regular teeth which they keep scrupulously clean and which afford few opportunities for the lodgment of food-particles, may fail to afford the evidence of a lead-line, even though they be saturated with lead. On the other hand, a lead-line once formed may persist, it is said, after recovery has taken place.

Without forgetting the exceptional conditions named, we may say that a blue line on the gums is a very sure evidence of lead poisoning.

The important point to bear in mind is that the absence of a lead line does not exclude lead poisoning.

Again, the onset of lead paralysis of typical form (paralysis of extensors of wrist and fingers, without implication of the supinator longus) in persons who have previously suffered from colic is a combination of circumstances which justifies us in regarding the case as one of lead paralysis, even in the absence of a "lead line," and in the further absence of any assignable cause.

Even if there be no lead line, and if the paralysis be not of a typical kind, we are justified in assum-

ing lead to be the cause of the trouble if the patient's occupation is one which brings him into intimate and daily contact with poisonous salts of lead.

A case of great interest in this connection was under my care in 1894.

The patient, E. S. H., æt. 35, a packer of furniture, was admitted on September 27th.

He had previously been admitted twice under Dr. Ringer, on July 5th and August 18th, suffering from abdominal pains, constipation, and albuminuria, and on both occasions he left the hospital cured of his abdominal trouble, and with his albuminuria reduced to a "trace" of albumen.

When admitted under my care he was again suffering from abdominal pain, vomiting, and constipation. His urine was of low specific gravity (1.012), containing one-third albumen, and there were superadded paralytic symptoms, affecting mainly the arms.

On electrical examination we found that the deltoids gave degenerative reactions, and were absolutely powerless, so that the patient could not raise either arm to the level of the shoulder.

The extensors of the wrist and fingers responded only to the strongest faradic currents, while the flexors of elbow, wrist, and fingers, and also the supinators longi and the interossei muscles responded normally to faradism, but were obviously paretic.

The patient complained of pain in both arms, and the nerve trunks were everywhere tender, especially in the axillæ. There was some impairment of sensibility to pains, but tactile sensibility appeared normal. The legs were paretic, but there was no actual paralysis. The patient walked fairly well, but feebly, and the reflexes were normal. There was no lead line, and the teeth were exceptionally clean. There was a distinct history of two attacks of typical gout during the previous year. The patient had been a moderate beer drinker, but he had not the aspect of a drunkard, and he persistently denied that he had ever taken alcohol to excess. He had drunk tea rather to excess. He made a fairly good recovery, and, after a sojourn at Eastbourne, and a few weeks at the Mineral Water Hospital, Bath, he returned to his work.

At the end of June, 1895, he paid his fourth visit to the hospital, but on this occasion his

symptoms were mainly abdominal, and there was no marked recrudescence of his paralytic troubles. The urine, however, was still of low specific gravity, and contained a trace of albumen.

Here, then, was a case in which there was no lead line, and in which there was no obvious risk of lead poisoning in the patient's occupation or surroundings, but which, nevertheless, we have felt justified in regarding as one of lead paralysis from neuritis. The justification of this diagnosis is found in—

1. The form of the paralysis, which is more typical of lead than of any other form of multiple neuritis.

2. The absence of other causes of multiple neuritis. It is true we have never discovered the cause of the lead intoxication in this case; but of all intoxications which give rise to neuritis, lead is apt to be the most insidious and the most difficult to discover.

3. The history of four attacks of colic, one of which immediately preceded the onset of the neuritis, give a very strong support to our diagnosis, because recurring attacks of colic without evidence of any cause of permanent intestinal obstruction, and without rise of temperature or distension of the abdomen, are extremely characteristic of lead poisoning.

4. The history of two attacks of gout, and the occurrence of albuminuria tending to become permanent, also lend support to the diagnosis of lead poisoning.

5. Finally, his recovery in hospital on four different occasions, and the immediate recrudescence of his troubles when he returns to his business and domestic surroundings, is a circumstance which points almost conclusively to the fact that his troubles are toxic, and that they are not autotoxic. Therefore the probability that they are caused by lead is very great.

One of the points in connection with lead poisoning, which has been much debated, is the seat of the lesion which produces lead paralysis, and while some have placed the lesion in the spinal cord, others have contended that the symptoms are compatible with a neuritis.

The fact that the paralyzes are generally symmetrical might cause one to infer that the lesions are spinal, but a little reflection will serve to convince us that arguments based upon this

ground are untenable. I may remind you that undoubted anterior poliomyelitis—spinal or infantile paralysis—is more often unilateral than bilateral in distribution, and that the undoubted neuritis which occurs from alcoholic, or as the result of diphtheritic, poisoning is almost always bilateral and symmetrical; in short, that while some undoubted spinal conditions are unilateral, some undoubted neurites are bilateral. The poison with which we are dealing circulates in the blood, and there is no more difficulty in assuming that corresponding nerve twigs on the two sides of the body suffer than there is in assuming that both anterior cornua at the same level should be attacked. Again, there is no more difficulty in assuming that certain nerve twigs are attacked than there is in assuming that the motor cells of the cervical enlargement at a certain level are selected for attack in preference to those above and below them.

If we admit that certain cells of the body stand in relation to certain poisons in the position of preference shareholders—which seems to be an undoubted, although an inexplicable fact—we must concede that such preference may be manifested towards cells at the periphery equally with those situated at the centre. If we accept so much then there is no difficulty in admitting that most nerve twigs supplying muscles may suffer in preference to nerve twigs and end organs conveying impressions from the skin.

One must admit that it may be impossible, in certain cases, to say whether the paralysis and wasting of a muscle be due to destruction of a group of cells in the anterior cornu or destruction of or pressure on the axial cylinders of certain nerves, or whether the second condition has followed as a consequence of the first.

The presence of pain in the limb, combined with tenderness of nerve trunks and muscles, undoubtedly points towards neuritis.

I think, however, that that which should make us regard lead paralysis as due to implications of nerve twigs rather than motor cells is the fact of recovery. A large number of cases of lead paralysis, if the cause be removed, get perfectly well; and as it is an admitted fact that the regeneration of a motor nerve is common, while the regeneration of motor cells, if it ever take place, is excessively rare, this fact of recovery may

almost be regarded as excluding much damage to the anterior cornua.

But it is not reasonable to assume that lead or any other poison has an exclusive preference for any particular organ or area, and one must be ready to admit that lead may inflict a damage upon the spinal cord or brain just as it does upon motor nerves, kidneys, liver, or intestines.

If we come to the conclusion that any case of recurrent abdominal trouble or paralysis is due to lead, then, assuming this conclusion to be correct, the prevention and treatment of the condition is within our grasp if we can succeed in detecting the source of the poison. The insidious and dangerous effects of lead seem to have been known to the Romans, and Vitruvius, the great Roman writer on architecture, mentions the danger of using leaden pipes for the conveyance of water. It is to be presumed, therefore, that the knowledge that lead-poisoned water will produce colic is very old. Vitruvius was also acquainted with the use of "red lead" for a pigment, but I have been unable to discover who first pointed out that painters who used lead paints were liable to colic. From the sixteenth century downwards descriptions of colics which were endemic in certain countries have been given by various physicians. One of the first writers was Francis Citois, a native of Poitou, and physician to Cardinal Richelieu, who described the *colica pictorum*, or the colic of the inhabitants of Poitou. Please note that the word *pictorum* has no relation to pictor, a painter. There is no evidence that Citois knew anything of "painter's colic" and he attributed the colic of Poitou to the acidity of the local wine. Similar endemic colics had been described in Normandy among the cider drinkers, and in this country an endemic colic was described as occurring among the cider drinkers of Devonshire, first, by Musgrave in 1703, and, secondly, by Huxham, who in 1739 published a treatise, "*De Morbo Colico Damnoniorum*." These writers had no notion as to the real cause of the colic, as a reference to the pathology of Huxham will show. Huxham attributed the colic to the acidity of the cider, and he says—

"By long and frequent drinking of a liquor of this kind, such a quantity of crude, gross tartar is thrown into the blood that it thence becomes very acrid; and not only the blood but, from that

impure source, all the humours thence secreted. So that, instead of a very soft lubricating mucus, separated by the glands, discovered by Dr. Havers, we have, as it were, a sharp coagulated matter, whence arises great pain in the joints and impotence of their motion. Instead of an exceedingly soft lymph to moisten the nerves, a corrosive ichor; and hence epileptical attacks. . . . At length, even the very bile, that variously useful balsam of the body, becomes corrupted and quite enervated by the superabundant apple-acid, though in its natural state it was designed to correct acidity."

I give you this as a very good sample of the pathology of 1739, but you will observe there is no mention or hint of the true cause of the Devonshire colic.

It was not until 1767 that Sir George Baker conclusively proved that the Devonshire colic was caused by lead poisoning in a masterly communication to the Royal College of Physicians, on June 29, entitled an "Essay concerning the Endemial Colic of Devonshire." Baker showed—

1. That acid drinks are not necessarily the cause of colic.

2. That the cider drinkers of Devonshire suffered more than the cider drinkers of Gloucester, Hereford, and Worcester.

3. That lead entered largely into the composition of the cider-presses of Devonshire, but not into those of the neighbouring counties.

4. He finally demonstrated that 18 bottles of Devonshire cider taken from his own cellar contained over four grains of metallic lead.

Not only in Poitou and Devonshire did these endemic colics get a footing, but in tropical countries as well. The "dry belly-ache" of the West Indies, and of some of the Southern States of North America, was thought to be a climatic disease until it was shown to be due to the contamination of rum by lead. Madrid was much troubled with an endemic colic, as were also the sailors in the French navy, but both these "endemics" have now been shown to be caused by lead poisoning; that in the French navy being attributed to the use of lead in the apparatus for distilling water. I may further remind you that in the old Bills of Mortality, issued by the parish clerks of London, "gripping in the guts" was a common cause of

death, and there can be little doubt that a fair proportion of these were cases of lead poisoning.

I mention these facts in order that we may appreciate the services of Sir George Baker. By showing conclusively that Devonshire colic was really lead colic, he gave us the power of arresting all the so-called "endemial" colics, and thus saved countless lives, and, what is far more important, prevented the crippling of useful lives.

Now that the symptoms of lead poisoning are well understood, and we are thoroughly alive to the dangerously insidious qualities of the poison, the question of "lead" is sure to cross the mind of the physician when confronted with cases in which the suspicion of lead is warranted.

In one of the cases which I have brought before you the suspicion of "lead" is so strong as to amount to a positive conviction in my own mind; but we have never been able to demonstrate the source of the lead, and in that important respect our duty towards the patient remains to be completed.

As regards the toxicity of the various preparations of lead, it is admitted that the salts of lead are far more dangerous than the metal. It is, indeed, a question whether metallic lead is to be regarded as a poison.

I remember hearing the late Mr. Hulke make allusion to the case of a gamekeeper who was troubled with that form of dyspepsia which, on account of the accompanying flatulence, the uneducated masses frequently characterise as the "rising of the lights." Whenever this gamekeeper was troubled with the "rising of the lights" he took a charge of shot to, as he said, "keep 'em down." This gamekeeper is said never to have suffered from saturnine intoxication; but we must remember that metallic lead must be liable to be converted into a lead salt by the acids of the stomach or by the acids taken with the food. The doctrine that metallic lead is not poisonous is, in all probability, a false doctrine for practical purposes, and in fact I was informed a few weeks ago, by Dr. George Oliver, of Harrogate, of a patient whose toxic symptoms were caused, he believed, by the practice of putting a shot in his mouth and idly chewing it.

By far the most important manner in which lead gains access to our bodies is by the con-

tamination of drinking-water. The result of such contamination is that lead is taken whenever the water is drunk, or whenever hot infusions made with the water are drunk, or whenever soup or food which has been made with or cooked in the water is consumed. If, therefore, the domestic water supply be contaminated with lead (no matter how slight such contamination may be), some of the dwellers in the house are sure to suffer eventually.

Not all waters dissolve lead. One of the advantages of a hard water containing lime, such as we use in London, is that it has little or no solvent power upon the leaden water-pipes and fittings. When we get our new supply of soft upland water from the Welsh hills we may have to alter our water fittings, but not till then. The conditions which favour the solution of lead in water are not completely understood, but it may be taken as an approximation to the truths that the purer and softer the water, the greater is its lead-dissolving power. Rain-water has great solvent power on lead: and in houses which are dependent upon rain for their water supply the greatest care must be taken to avoid the use of lead for water-pipes and gutters, storage tanks and service pipes. Waters which are acid in reaction seem to dissolve lead with a most dangerous facility, and the most recent investigations seem to show that the lead-poisoning which has occurred in some of the Yorkshire towns during the last few years is caused by the acidity of some of the peaty waters with which they are supplied.

I have no intention of taking up your time by cataloguing all the possible sources of lead-poisoning. Suffice it to say, that the lead may be absorbed from the alimentary tract, the respiratory tract, or the skin, and that the economic uses for lead and its salts are very numerous. Acid liquors must not be kept in leaden vessels, or in earthenware vessels finished with a lead-glaze, as we have learnt from the study of cider colics. Where lead poisoning is suspected, you must closely scrutinise the sources and surroundings of all food and drink. Food wrapped in lead, such as tea; canned or tinned foods, hermetically sealed with a leaden solder; soda-water syphons with leaden fittings; tobacco wrapped in lead foil; snuff adulterated with chromate of lead; anchovy paste, or lozenges coloured with the same material, are a

few among the many recorded instances of accidental lead poisoning by food.

That lead can be absorbed by the skin seems also to be established, and not a few cases of lead poisoning have resulted from the use of hair dyes containing lead, or from the application of pigments containing lead by theatrical artists for the purposes of "making up."

Lead poisoning is common to many trades—as the manufacturers of "white lead," and other lead pigments, colour-grinders, painters, pottery-glazers, makers of glazed cards, etc.; but it seems clearly established that those who are engaged in the manufacture of lead-products, such as pigments, are more liable to suffer than those who are engaged in manufacturing articles from the pure metal.

I have said enough to show that lead is almost omnipresent, and that when you have reason to suspect lead poisoning, the source of it often requires diligent search, in which the assistance of a skilled chemist is necessary. One word of caution. A medical friend of my own, practising in the country, was confronted with several scattered cases of lead poisoning, and for a very long time he failed to discover the cause; but finally he found it, as he thought, in certain packets of an article of consumption which was being rather freely distributed in his district. Lead was actually found, on chemical analysis, in the article contained in these packets, and, armed with this knowledge, my friend, acting as every doctor should do, in the best interests of his patients, gave a warning that the article in question was unfit for consumption. Thereupon the proprietors threatened him with an action for libel; and, although the action was settled out of Court, it was the source of considerable worry and expense while it was pending.

I take it that the proper course to pursue in such a case would be to impart your suspicions to the sanitary authority confidentially, and allow them to deal with the matter, with the assistance of their analyst.

Finally, I have to speak of the treatment of lead poisoning.

Our first duty is, if possible, to discover the source of the lead and stop its administration, and no more need be said on this head.

But it often happens that it is impossible to

change the occupation of the patient, and when that is the case we must adopt such measures as may commend themselves for lessening the risks of such occupation. The chief of these are cleanliness and ventilation. A painter or any other worker in lead should be scrupulously careful to wash his hands before eating, and to thoroughly cleanse his hair, body and mouth every day. He should always work in washable overalls, and a clean suit should be put on at least once a week. If this be done the worker with lead pigments runs far less risk than if he inhabits for months together the same dirty paint-bedaubed suit of clothes, and is content to eat his meals with paint-bedaubed hands and to wear a mass of dirty hair. A moustache, if it be properly washed and cleaned, is probably a protection to the worker in lead.

Good ventilation is essential, and I would remind you that one of the patients, whose case I have related, attributed his attack to working in an unventilated room in hot weather.

Workers in lead must be well nourished. Dr. Thomas Oliver, of Newcastle, has made experiments which show clearly that saliva, gastric juice, and bile, are all capable of dissolving lead, but that if starch be mixed with the lead, less of the lead salt is dissolved by the saliva; if proteid be mixed with the lead, less of the lead is dissolved by the gastric juice, and that if fat be mixed with the lead, less of the lead is dissolved by bile.

The conclusion is, then, that saliva, gastric juice and bile have no preference for lead in the presence of those alimentary principles upon which these three digestive fluids are designed to act. The well-fed lead-worker runs less risk than the semi-starved, ill-fed worker, and the man who goes to his work with a stomach full of food is less likely to suffer from lead poisoning than he who goes to his daily toil on an empty stomach. Dr. Thomas Oliver is fully supported in this conclusion by the experience of the manufacturers of lead products.

Dr. Oliver is of opinion that all lead workers should be total abstainers from alcoholic drinks, and this conclusion is supported by the well-known liability of lead-workers to suffer from kidney disease and gout.

As a prophylactic it has been the custom, in some lead works, to provide the employés with a

"lemonade" made with dilute sulphuric acid instead of lemon juice, with the object of converting the lead in the alimentary tract into the comparatively insoluble lead sulphate. Dr. Oliver supports this practice, but it must not be forgotten that lead poisoning has resulted from taking lead sulphate, and that the constant administration of dilute sulphuric acid is apt to produce constipation, which, in the lead worker, is very undesirable. It is stated, also, that when this acid drink is provided it is difficult to get the workpeople to take it continuously.

To get rid of the poison from the system and overcome the lead colic, the continued administration of magnesium and sodium sulphate, combined with potassium iodide, has given the best results. It is a good plan also to give the saline aperients mixed with hot water. Enemata may be employed with advantage. Belladonna is also a useful adjunct to the salines.

Lead is eliminated from the system by the bowels and the kidneys. Dr. Dixon Mann is of opinion that when lead is administered medicinally by far the greater part is eliminated by the bowels. Thus he gave two grains of acetate of lead to a patient on five consecutive days. On the fifth day the fæces yielded the equivalent of five grains of lead, and on the sixth day four grains, while one milligramme of lead was the largest amount eliminated by the kidneys on any one day. Whether the lead eliminated by the fæces had simply passed through the alimentary tract is doubtful, but I would remind you that arsenic, antimony, and mercury appear to be excreted by the large intestine after absorption from the higher parts of the alimentary tract, and it is possible that the same process may take place in lead poisoning.

In Dr. Oliver's cases the largest amount of lead eliminated by the kidneys in a single day was .17 grains, and the lowest amount recorded was .002 grains. The daily eliminations fluctuated in a remarkable way, and occasionally the elimination of uric acid and lead appeared to bear an inverse ratio to each other.

It has long been the practice to administer potassium iodide (gr.ij to gr.v) in cases of lead poisoning, and Dr. Oliver supports this practice; but it may be stated that Dr. Dixon Mann has failed to find that this drug increases the elimination by the kidney. Iodide of potassium is

frequently administered with apparent success in cases of sclerosis and neuritis, no matter from what cause: and, seeing that lead produces sclerotic changes in a most remarkable way, I think we are justified in continuing to administer potassium iodide in moderate doses.

Warm baths are of undoubted service in lead poisoning. They were in repute more than a century ago, and sufferers from Devonshire colic found benefit from the warm baths of Bath long before Baker showed that this colic was caused by lead. It is probable that soaking in warm water stimulates nutrition and metabolism, and there seems to be no doubt that it is useful in lead poisoning. Sulphur baths have enjoyed a great repute, but whether they are more efficacious than the "indifferent" water of Bath is doubtful.

For the treatment of the paralysis the most important thing is to keep the limbs protected and warm. Warm baths are very useful, and a man who cannot move a paretic limb in the air may manage to do so when it is immersed in water, which helps to buoy up the limb and diminish the work of the muscles.

Massage and electricity are both useful in the treatment of lead paralysis, but neither of these agents must be employed so long as the nerves and muscles remain tender. If either of these agents be used in the painful stage of the paralysis they are apt to do harm rather than good. One of our patients was subjected to this treatment when the nerves were tender. The right arm and shoulder were daily rubbed by the clinical clerk, while the left arm and shoulder received no such treatment, and it is certain that in this case the left arm (which was not rubbed) seemed to recover power more quickly than the corresponding limb.

While the nerves are tender I think it is a good plan to apply strips of capsicum plasters along the line of the tender nerves. These serve as a protection, and act also as a mild counter-irritant.

When the patient begins to improve, he is benefited by the administration of tonics, such as liquor strychninæ, combined with mineral acids. The diet should be light and nourishing, and finally the patient should be sent to a convalescent home before returning to his work.

CLINICAL LECTURE

ON

STRICTURE OF THE RECTUM.

Delivered at University College Hospital, Dec. 9, 1895.

By CHRISTOPHER HEATH, P.R.C.S.

GENTLEMEN,—We have at the present moment in No. 5 Ward, two cases of stricture of the rectum. The patient about whom I shall first speak is a woman, æt. 35. She states that she has had good health, but allows that she has had two miscarriages. Further than that we can get no specific history. I mention this at once because she is suffering from stricture of the rectum, which is generally thought to be due to syphilitic poison. About seven years ago (in 1889) the patient experienced great difficulty in the passage of the motions, much straining being required in order to defæcate; the stool invariably contained a bloody discharge, and great pain was felt during the process. At first no notice was taken of this, as she thought it was due to piles. In both the patients whom we are considering to-day the symptoms were thought to be due to piles, in one of them the opinion being shared by the medical attendant. I would therefore advise you never to treat a case of so-called piles without first examining the rectum. This woman of 35 has been suffering from difficulty in defæcation for seven years, and four of these were allowed to pass before she sought medical assistance. In 1892 she went to Guy's Hospital, where she was informed that she was suffering from stricture of the rectum, and was advised to enter the hospital. She made arrangements to do so, but for various reasons did not then become an in-patient. Her condition remained the same until last June, when she went to the Lewisham Infirmary, but only stayed there two weeks. There she was told she would have to go to a hospital, and about a month ago she came here, and was admitted into No. 5 Ward on October 28th.

Here, then, is a woman of only 35 years of age, whose rectum is practically completely blocked. Those of you who examined her the first time she was seen after admission, will remember that there was an exceedingly small opening, which would

hardly admit the end of the little finger. When I found the opening was so extremely small, I ventured to insinuate my finger into it with a little force, which dilated it sufficiently for me to feel that though the lower part of the rectum was exceedingly rough, evidently the seat of extensive disease, yet when I passed my finger through the tight ring I found healthy mucous membrane above. There is no obvious history of syphilis in the case; the only fact which points in that direction is that she has had two miscarriages, and we all know that miscarriages may be due to other causes than syphilis. Then arises the question, What is the nature of the disease? Is it certainly syphilitic stricture? That is the opinion of most surgeons. They think that these young women have syphilitic stricture, due to infiltration of the walls of the rectum; that they have acquired syphilis early in life, say, when they are 20; that then they go through more or less secondary symptoms, and that when they arrive at about 30 years of age they begin to develop gummata in the rectum. This fact is somewhat remarkable, because, as you are aware, gummata in other parts of the body do not usually show themselves so early in life. We look upon gummata as some of the later manifestations, certainly the tertiary manifestations of syphilis, and we find gummata on the tongues of people over 50; but it is unusual to find gummata at the age of 35. There are other views, however, put forward about these cases. One of them is that it is not syphilis at all, but is a chancrous ulceration, extending from the vagina. These women are, of course, very apt to get various forms of disease about their genitals; and it is suggested that when they get soft sores upon the labia or in the vagina, the discharge from these sores is apt to inoculate the anus, spread up the rectum, and so give rise to the stricture. I must say that this has always seemed to me a very unsatisfactory explanation.

I thought I had better look up one of the most recent books on the subject of syphilis, namely, Mr. Alfred Cooper's work, the second edition of which is published this year, and is edited by Mr. Edward Cotterell, who was an old house-surgeon of my own. He says:—"Gummatous deposits and infiltration of the submucous tissue are the most common causes of syphilitic ulceration and stricture of the rectum. Fournier,

however, dissents from this view, and thinks that, in the majority of cases of stricture of the rectum in syphilitic subjects, the condition is due to an infiltration of the ano-rectal walls with a new formation of unknown structure, a hyperplastic proctitis, which generally goes by the name of ano-rectal syphiloma, and which has certain resemblances to the fibroid degeneration seen in the nose and larynx. It undergoes fibroid changes, and produces contraction of the calibre of the bowel. He states that, as a general rule, neither ulceration nor cicatrization can be found in the rectum in cases of syphilitic disease, and hence he infers that the morbid changes involve the submucous tissues rather than the mucous coat. Zeissl, on the other hand, states that the lesion is due to gummatous infiltration of the submucous tissue. The disintegration of the deposit causes ulceration of the free borders of the longitudinal folds and of the mucous membrane between adjacent folds. When cicatrization takes place, some of these folds are either obliterated or become adherent to each other: in either case the calibre of the bowel is reduced. Zeissl further states that distinct gummata are sometimes developed in the submucous tissue, and are followed by disintegration, ulceration, and severe stricture. He reports a case of this nature occurring in a man, presenting also syphilitic nodes and sarcocele." You will see, therefore, that there is a difference of opinion; but I think we may say that the weight of authority is in favour of there being gummatous deposit in the walls of the rectum. The only point which at all favours the view that there may be some infiltration from the vagina to the rectum, is the notorious fact that these strictures are infinitely more common in women than in men.

Be the cause what it may, there can be no question how the disease comes on—that there is first a deposit, and then ulceration, and that during ulceration there is profuse discharge. The woman who is now in the ward has no special discharge from the rectum, but if you had seen her some time ago, it would have been evident. In recent cases a profuse, semi-purulent, offensive discharge is present. Moreover, there is about the anus a feature which is very characteristic of this syphilitic ulceration: that the skin becomes hypertrophied, causing those loose "tabs" of skin which I have

mentioned to you before. This was so evident in the woman in the ward, that on seeing them I ventured to say we should be pretty sure to find syphilitic stricture.

When you see these cases in the ulcerative stage, you must heal up the ulcers, and at the same time prevent undue contraction. During that stage, I think there can be no doubt that antisyphilitic remedies—particularly iodide of potassium—do good. Syphilitic ulcer of the rectum should in fact be treated much in the same way as syphilitic ulcer of the leg, viz., by giving iodides internally, and applying a local mercurial. It is very much easier to apply an ointment than lotions to the rectum; therefore the best plan is to let a patient who is suffering from syphilitic ulceration of the rectum have some sort of bougie with which she cannot do herself any harm, smear it with a little blue ointment, and have it pushed up every night. This brings the ointment into direct relation with the sore; moreover, the passage of the bougie tends to keep the passage dilated. You ought, of course, to carry the treatment on for some time, but my experience of this class of patient is that they will not carry out any treatment properly. As soon as they begin to get a little better, and the ulcer commences to heal, they stop the treatment, and do not come again until they get into very much the same condition as our patient in the ward, *i.e.*, until they have an almost impassable stricture. Fortunately, the stricture is always low down, within reach of the finger, and this enables you to dilate it if you are sufficiently careful.

For that purpose there is no better bougie than an ordinary old-fashioned tallow-candle which has been previously greased. They run about eight to the pound. A piece of string can be tied to the wick at the end, to prevent the candle going out of reach. Remember that these candles can hardly do any harm, but I regret to say that only in September last I distinctly brought about the death of a woman by using a bougie upon her—a case very similar to the one in the ward, but not quite so bad. I had her down in the theatre, gave her chloroform, dilated the stricture up with my finger (I don't think I passed a bougie at all), and I told the house-surgeon not to pass a bougie, for fear of any accident. She survived the operation, and no harm appeared to have been

done. When I next went round the ward I, without any difficulty, passed one of the ordinary small bougies, taking the precaution to have it warmed and well greased before use, and leaving it in only an hour. The patient made no particular complaint, but within a few hours after the withdrawal of the bougie she began to get urgent symptoms of pain in the abdomen, which went on to acute peritonitis, and she died. Then we found, as I had anticipated, that the bougie had passed through the stricture, impinging upon the softened part of the bowel above it, and had penetrated into the peritoneum. That is by no means a solitary instance of the kind in surgery. Of course each such case causes great regret, and the occurrence should be guarded against as much as possible.

I show you an instrument here merely for the purpose of warning you against using it, viz., a very powerful dilator, which was invented for these strictures by a surgeon now deceased—Mr. Armstrong Todd. You will notice that when I turn the handle there is an ingenious arrangement of cross-bars by which the two blades are widely separated. When this instrument was first brought into use it was thought to be exceedingly good, and surgeons employed it somewhat freely. The first inconvenience they found was that, when shutting the instrument prior to withdrawal, the mucous membrane was very apt to be caught between the blades, and torn in withdrawal. To obviate that, the use of an india-rubber cover, resembling a long finger-stall, was suggested. By this means all the good points in the instrument are retained, and the bad avoided. But the danger is this: if you dilate a stricture suddenly and forcibly, the tear is apt to run further than you imagine, and you are in danger of going through the rectum and into the peritoneum. Cases are met with in which the contraction is exceedingly strong, and the stricture hard and tough, so that little progress can be made with the ordinary bougies. In such cases the best plan is to notch the stricture with a knife; it is very much safer than tearing. With your finger introduced into the stricture you slip up a blunt bistoury, and you can then, by using a little pressure, notch the stricture in three or four places. This, however, should only be done with patients who are under your control; never do it to an out-patient, for fear of anything going wrong. The worst of these cases is that the

disease is very apt to recur, owing to the fact that the mucous membrane is practically destroyed, and nothing is left but a mass of fibrous tissue, which is very apt to contract. You may do good for a time, but the patient, if she have any confidence in you, will come back from time to time saying she is as bad as ever. Perhaps the best course with these patients is to perform proctotomy, by which I mean the division of the coats of the rectum on the posterior surface, where there is no peritoneum, going right down to the sacrum. It is not such a severe operation as it might at first appear. The tissues are all indurated, there is very little hæmorrhage, and what there is can be controlled by temporary plugging. This will often benefit your patient more than any other proceeding.

The only alternative in these very serious cases is to do colotomy. I have, in former years, done this for cases of this nature, and have seen it done many times; but I must say I do not recommend it. Colotomy makes the patient an invalid for life. These women are generally under 40, and if I were to do colotomy for the woman in the ward, she would be a confirmed invalid before she has reached middle life. Therefore I think one should exhaust every other means before recommending colotomy. Of course, if a patient comes to you in whom the contraction of the bowel has been allowed to go on until a state of distension is produced, it may be necessary to do colotomy as the only means to save her from dying of obstruction; but with this exception, after having had some experience of the operation, I should be inclined not to resort to it.

We now come to the other form of stricture, which, I am sorry to say, is also very common—viz., epithelioma of the rectum. You will remember that a few days ago there was an old lady in the hospital whom we all examined, and who undoubtedly had epithelioma of the rectum. But the growth was only on one side, the vaginal side, and there was no particular difficulty in the fæces passing, and there was no ulceration to give her much pain. I, therefore, advised her to wait, saying I would take her in at any time and operate upon her if the symptoms became more urgent, but that on the whole she could very well wait, and see how she got on.

Here is a very different case, in a woman of

only 50. She first noticed something wrong in the rectum last spring, and was, for a time, treated for piles. Five weeks ago she was examined, and told that there was something more than piles. She has had great difficulty in passing motions, and that difficulty has increased up to the present time. The state of the patient is now as follows:—She is pale, looks considerably more than 50, rather cachectic, complains of difficulty in getting the bowels to act; there is some discomfort in the abdomen, not actual pain. The abdomen is not distended, but is quite flaccid. Nothing abnormal is felt *per anum*, but the lower part of the rectum is much dilated, and contains some fæces. Three inches from the anus there is an irregular nodular growth projecting into the lumen of the gut, and causing so much narrowing that the finger can only just be inserted. The growth is annular—it forms a complete ring in the bowel; it is very hard, but no ulceration is felt. Examination causes pain, but not bleeding. The finger cannot be passed through the growth, therefore its extent cannot be determined. *Per vaginam*, a hard mass is felt at the upper part, projecting into and invading its wall, one nodule being close beneath the mucous membrane in the posterior fornix. Although we are not able, in this particular case, to exactly determine the extent of the growth, we know that it is already involving the vaginal wall, which makes it pretty evident that the disease is far advanced. Unless I do something to relieve the condition of things, in a short time she will probably have absolute obstruction, and die from distension.

In this patient the disease has existed for many months; but you must be prepared to see such cases in the early condition when in private practice. Cancer of the rectum affects men as frequently as women, and in the early stage the man has perhaps noticed that he has not been feeling well, has been generally out of sorts, and has perhaps had some indigestion. But the only definite symptom he has noticed is probably the occurrence of morning diarrhoea. Now, that is very characteristic. The patient finds that he cannot get through his bath and his dressing without going to the closet. After he has breakfasted his bowels are again opened, and after the stool they are still a little irritable. He perhaps evacuates again at night, but it is the morning diarrhoea

which is important. If he takes the trouble to look at the motion, he will see that it is broken up into small pieces; it is not the uniform mass of fæces which is met with in health. When you hear such a story you should always suspect that the patient may have trouble about the bowel. I may say that the late Sir Andrew Clark, to my knowledge, "spotted" a good many cases of cancer of the rectum which had not been suspected, by always insisting upon examining the rectum when he found any symptoms pointing at all in that direction.

It is a satisfaction to tell a patient he is suffering from nothing serious. Therefore let me again beg of you to take every opportunity of examining healthy rectums, so that you may be able to detect the slightest deviation from health. You will find, in most cases of cancer, that somewhere just within reach of the finger there is a hard lump, perhaps on one side, possibly all round. Sometimes the lump is just out of reach of the finger; in such cases, by drawing gently upon the mucous membrane, you can bring it within reach. I advise you to get the patient to strain down, and a good plan is to give him an enema just before the examination, then let him go to the closet and get down all he can, and you will find that he has brought the disease within reach of the finger. You will remember this particular patient told us that when she went to the closet she felt something in her bowel come down. If the stricture comes down close to the anus, one finds the condition which was at one time laid great stress on, but which is, in reality, quite unimportant—the occurrence of tape-like motions.

If you make the diagnosis correctly, you must, of course, inform the patient or some friend what the probabilities are. I do not mean that you must tell a patient who has a lump in his rectum, or a slight ring, that he must have colotomy done; that would be ridiculous. But you must let somebody know that his health is not what it should be, and that he will probably get worse. Meantime, you will give general directions. In the first place, tell him to avoid all indigestible food; he may take chicken, fish, or lamb, rather than beef or mutton. There is no objection to puddings, but he should specially avoid all stimulating foods, such as spices, curries, and hot soup, which are apt to cause irritation about the lower bowel. He

should be very careful about liquor. He should avoid all champagne and effervescing wine; beer is not good, and there is nothing better than a little spirit and water in great moderation. Then he certainly ought to get into the habit of having his bowels opened at night, so that if there is any difficulty with them, he may be able to get eight or ten hours' rest afterwards. The best plan is to take some slight laxative—a little oil or liquorice powder, or sulphur—in the morning, to produce a mild evacuation in the evening. It is well also to throw up a little water and wash the bowel out, because the fæces irritate the surface.

Lastly comes the question of sedatives. If the patient has pain, which keeps him awake at night, it may be advisable to begin the use of suppositories, containing $\frac{1}{4}$ gr. morphia, or $\frac{1}{4}$ gr. belladonna, increased as occasion requires. While the patient can go about his work and get fairly comfortable evacuations, he should do so; the time will come when he cannot do it, viz., when ulceration commences. This is accompanied by discharge and excruciating pain, and generally patients have to lay up, and you are obliged to increase the quantity of morphia to produce any comfort at all.

Then arises the question of interference. For my own part, I have made up my mind that removal of cancer of the rectum by external operation is *not* advisable. I have done it, and seen it done a good many times, and it seems to me a most unsatisfactory operation, because, if you do remove the whole of the cancer, you are certain to get so much contraction afterwards that the last stage is worse than the first, and eventually colotomy has to be performed. Besides, the growth is often too far distant from the anus.

The operation which I propose to do for this woman next Wednesday is to open the sigmoid flexure of her colon in the left groin. There is an older operation, called Amussat's operation, which was in the loin, and it is a curious historical fact, which Sir John Erichsen mentions in his book, that he was present, as a student, in Paris when Amussat performed his first lumbar colotomy. Lumbar colotomy answers extremely well, but we have improved upon it by inguinal colotomy, although I still do lumbar colotomy in tight strictures where the bowel is full of fæces. In a case such as that of the woman now in the ward,

there is no doubt that inguinal colotomy is preferable. I shall make an incision about 4 inches long—2 inches above and 2 inches below the anterior superior spinous process, in a slight curve. I shall cut through the abdominal wall, the external oblique, the internal oblique, and the transversalis, or rather, as a modern improvement, I shall cut through the external oblique, and tear through the other muscles, thus making some sort of sphincter for the bowel afterwards. Having got through the three muscles, I shall come upon the fascia transversalis and the peritoneum, both of which have to be opened. This brings one, without the slightest difficulty, upon the sigmoid flexure, which is recognizable by the three longitudinal bands, which are unmistakable. If the sigmoid flexure is not actually in view, the small intestine has to be pushed inside and the flexure sought for. It is necessary to make sure that you are above the seat of the disease. It is not in every case that the disease can be felt from the rectum, and occasionally colotomy has to be performed without feeling anything. It has happened to me to have opened the left side, found that I was not above the disease; then to have stitched up the wound and opened the colon on the other side, the patient making a good recovery.

The colon is pulled up and brought out of the wound, and I am then in the habit of using a glass rod to push through the meso-colon to support the bowel in its place. Some surgeons do not use this, and I do not insist upon it; but I do insist on the necessity of stitching the colon, although I know some surgeons say it is quite unnecessary, and that if the parts are merely left in apposition they will unite. But you must remember that you have got the peritoneal cavity open, that the patient may have a violent attack of retching when he gets back to bed, and a piece of small intestine may be forced out by the side and produce a hernia. This actually occurred in a case of my own, where I had used stitches, the vomiting forcing out a couple of the stitches, and the small intestine coming through under the dressings. The house-surgeon thought it right to examine the parts after the retching was over, found what had happened, put the small intestine back, with two or three extra stitches, and no harm came of it. But in another case it might

not be found out directly. It was formerly the practice, and was usual when I did my first operations, to draw forward the parietal peritoneum, and attach it to the skin. Someone suggested that this was quite unnecessary, and Mr. Greig Smith, of Bristol, who is a great authority on these subjects, maintains that peritoneum and skin do perfectly well together, and that they unite without any trouble, and so it appears to be. The bowel, having been brought well out, is dusted with iodoform, and the dressing put upon it. It is well to leave it alone for four or five days if possible. I shall do the operation on Wednesday, and shall probably open the bowel when I go round on Monday. If the patient were uncomfortable, I should open it on Saturday or Sunday. The bend which you make in the bowel by the glass rod practically blocks the lumen of the bowel; and the patient will pass no flatus *per anum* after the operation.

When you have cut away the projecting surface of the bowel you have two openings, the upper one leading into the bowel, from which the fæces immediately project, and the lower opening which leads into the rectum, which is usually empty. If all goes well, the patient will begin to defæcate naturally from the artificial anus, and in the course of a fortnight or so all the parts cicatrize, and there is usually a wonderful amount of sphincter power, so that the patient is able to know fairly well when fæces are ready to pass, although such patients cannot generally retain the flatus. With regard to the upper opening, we generally adapt to it an india-rubber colotomy pad, the projecting finger-like process of which is put into the lower opening, and then the fæces emerge from the upper opening beneath the pad. The lower opening should be washed out about once a week. If this is not done, inspissated mucus collects, and is apt to irritate. A week or ten days after the operation I tell the nurse to put the patient on a closet stool, and wash out the bowel with warm water.

If, at the operation, there should be difficulty in finding the bowel, a very curious thing sometimes happens, viz., the bowel gets twisted, and when you come to open it you find that, instead of fæces coming out at the upper opening, they emerge at the lower one. I have heard of that a good many times, but have only once met it myself. Why it should be I cannot tell. The patient we have been

discussing looks as if she will do well. Of course, the operation I am going to do will in no way cure her rectum, but it will remove the source of constant irritation from fæces, and will entirely obviate any possibility of her becoming obstructed, and it will therefore prolong her life. In these cases the patients may go on very comfortably for two, or even three years, which is the average duration of life after colotomy; whereas, if the operation were not performed, she could not live more than three or four months, so that there can be no doubt about the propriety of operating in her case.

THERAPEUTICAL NOTES.

Corneal Ulcer.—Cases of intractable corneal ulceration, which have long resisted the usual methods of treatment, quickly improve under the use of glycerite of tannin and carbolic acid prepared according to the following formula:—R. Carbolic acid, 15 grains; tannic acid, 30 grains; glycerine, 1 drachm. M. Carefully cleanse the eye with warm sterilized water strongly impregnated with boric acid, introduce a few drops of weak cocaine solution to annul pain, and, after a wait of five minutes, freely touch the ulcer with the glycerite by means of a fine camel's hair pencil. Repeat twice or thrice daily for the first few days, and then gradually discontinue as healing progresses. In conjunction with this treatment it is well to maintain the usual instillation of atropine, Unpleasant reaction never occurs.

Medical Record.

Intermittent Fever.—Aufrecht having found, in common with many other authors, that tablets of quinine frequently pass through the digestive tract without being absorbed, advocates the use of neutral tannate of quinine for prolonged use. It has scarcely any taste, and, as it contains three times less quinine than the hydrochlorate, larger doses must be given. He has found the following formula very efficacious in inveterate cases:—R. Arsenious acid, $\frac{7}{8}$ grain; hydrochlorate of quinine, 31 grains; glycyrrhiza-powder, 46 grains; glycyrrhiza-juice, q.s. to make 30 pills. Two pills three times a day.

Ther. Monatshefte.

Digitalis in Pneumonia.—Nægeli-Akerblom regards digitalis as the most valuable agent in acute pneumonia when used in large doses. The drug, according to his investigations, causes hyperleucocytosis, both in man and animals. He advises hydrotherapeutic measures in connection with the digitalis.

Cent. f. inn. Med.

Subacute Bronchitis.—

R	Ammonium chloride		
	Sodium iodide	...	āā 3iij
	Syrup of Tolu	...	
	Syrup of senega	...	āā 3jss

If a spasmodic element be present, add sodium iodide, 2½ grains to each dose.

Philadelphia Polyclinic.

Hay Fever.—

R	Mentholis	gr. xx
	Olei amyg. dulcis.	3 ij
	Acidi carbolic	℥ x
	Cocain. hydrochlor.	...	gr. vj	
	Ung. zinci oxidi	3ss

Sig.: Apply thoroughly to the nostrils on cotton attached to a tooth-pick.

Medical Record.

Tetanus.—L. Oscherowski reports the cure of a case that has resisted morphine, opium, and chloral. Cure was effected by injections three times a day of twelve drops of a two per cent. solution of carbolic acid in water.

St. Petersburg Med. Week.

REVIEW.

Appendix to Medical Digest. By Dr. RICHARD NEALE.

Anyone who has once used Neale's Digest will not willingly relinquish the advantage offered by this Appendix (bringing the whole digest down to August, 1895), and we have, therefore, much pleasure in drawing our readers' attention to its publication. How Dr. Neale has found time to make the references is a puzzle to us; but for accuracy and for utility, Neale's Digest has already won for itself such a reputation that we simply accept the fact and leave the wonder. Every busy practitioner should certainly have the work complete if he wishes to know when and where information on any and every affection has been produced by others.

THE CLINICAL JOURNAL.

WEDNESDAY, JANUARY 29, 1896.

A POST-GRADUATE LECTURE

ON!

TUBERCULAR ARTHRITIS.

Delivered at the Hospital for Sick Children, Great Ormond Street, on November 28th, 1895,

By **GERALD R. BALDWIN, F.R.C.S.,**

Surgical Registrar.

OF all the cases treated in the wards of this hospital, probably 75 per cent. are of tubercular origin, and of these nearly 50 per cent. are instances of tubercular arthritis. On account of its frequent occurrence, I have thought no subject more deserving of your consideration than that of tubercular disease in joints. Of all the joints in children affected by this terrible scourge, the hip is most frequently attacked; next in order we find the knee, tarsal joints, elbow, ankle, shoulder and wrist. It will not be necessary to speak of the minute pathology of tubercle—with that you are all well acquainted; but I must say a few words as to the various ways in which a joint may be infected. The attack may come from within the joint or from the surrounding tissues. In the first case the synovial membrane is practically always the structure first infected; in the second the disease may originate in bone, in the soft tissues around the joint, or in a bursa in close proximity to the joint. If the bone be first infected, the site of predilection for the bacillus is the rapidly growing and therefore vascular tissue on the diaphysial side of the epiphysial cartilage. Another position in which the disease may originate is the young bone just beneath the articular cartilage. If the tubercular infection spreads from the soft tissues around a joint, it most commonly begins as a chronic abscess which perhaps bursts into the joint, or else each structure separating the abscess from the joint cavity is in turn invaded, until the synovial membrane becomes infected. As I do not wish to obscure the subject, I will here digress for a

moment in order to point out that disease beginning within a joint may be associated with an extra-articular abscess. In such a case the disease spreads from within outwards. The presence of a true extra-articular abscess, meaning thereby one in which the capsule remains intact, is less common than frequently supposed. The diagnosis of extra-articular abscess is often made when suppuration has in reality begun within the joint, with subsequent destruction of the capsule and escape of pus into surrounding tissues. For after its escape the pus may track and point a long way from the original site of rupture, and the communication with the joint escape detection. I have already mentioned three forms of abscesses in the neighbourhood of joints, and must now point out a fourth. Occasionally tubercular disease primarily infects a bursa, setting up a chronic bursitis, the products of which later on tend to undergo fatty degeneration and possibly suppuration. From the bursa the neighbouring joint is readily invaded. Having pointed out the different situations in which tubercular arthritis may originate, I shall next consider where it most commonly does begin in the various joints of the body. In the hip joint the disease most commonly commences in the neck of the femur, in the neighbourhood of the epiphysial cartilage, or less commonly beneath the articular cartilage. From the latter site its spread into the joint has little to hinder it. In the neighbourhood of the epiphysial cartilage the deposit of the bacillus is soon followed by an abundant round-celled infiltration, which presses upon and gradually absorbs the soft cancellous bone around it until that bone is replaced by a new granulation tissue in which some, though commonly but few, giant cells are seen. The subsequent progress of the disease may be towards recovery, and we frequently see cases of hip disease treated early by complete rest to the joint recover perfectly. In such cases the round cells are in turn replaced by plasma cells from surrounding connective tissues, which form firstly fibrous tissue and ultimately true bone. Unhappily, however, either from lack of treatment, improper treatment the result of

mistaken diagnosis, or even in some cases despite careful treatment, the disease advances a step further; the round cells undergo fatty degeneration and suppuration ensues around the caseous patch, and we have an abscess in the neck of the femur. Such an abscess is placed in the most favourable site for secondary infection of the joint. The upper epiphysial line of the femur lies entirely within the capsule of the hip joint. On the joint side the abscess meets with least resistance, and that is the direction it tends to follow, ultimately extending into and causing secondary infection of the joint cavity. It may reach the joint by spreading along the epiphysial line, and cause separation of the epiphysis, which is in such cases, if excision be performed, found as a sequestrum within the joint; or it may gradually destroy the head of the bone, and thus find its way into the joint. In some cases the disease spreads towards the diaphysis, and does not invade the joint, but unfortunately such a result is a rare one. Although this paper is intended to treat mainly of the pathology of tubercular arthritis, it seems to me that I should not entirely neglect the clinical aspect of the disease, especially in dealing with so important a division of the subject as tubercular osteitis originating in the upper articular end of the femur. For if we can diagnose the disease early we may hope by skilful treatment to prevent its spread to the joint. It is unfortunately very rarely that we are afforded an opportunity of doing this. As a rule parents neglect to place a child under medical treatment before the joint is already implicated. A slight limp and occasional pain are frequently disregarded, and medical advice unsought before hip disease causes symptoms too obvious to be neglected. Yet there are certain signs by which it is sometimes possible to separate tubercular osteitis near a joint from disease in the joint itself. Of all joints this is most difficult in the hip, the articulation is deeply buried in muscles which obscure the signs due to distension of its synovial cavity, while the epiphysis is situated within the capsule, and thus tenderness resulting from pressure upon the affected bone cannot well be distinguished from tenderness the result of pressure upon an inflamed joint. However, the following signs may at least aid us in tubercular osteitis: freedom of movement in the joint, although the involuntary

limp points to disease in its neighbourhood; pain elicited on bringing the bony surfaces into contact, as by pressing the head of the femur firmly against the acetabulum, jarring the foot, or forcibly abducting the limb; and absence of that early fulness in the groin arising from distension of the synovial cavity, which is present from a very early stage in the primarily synovial form of disease. Of the treatment I have nothing to say, except that if we have clear evidence that the disease is as yet extra-articular, we should in operating attack it from outside the joint, and thus attempt to eliminate the mischief without interfering with the functions of the joint itself.

In some cases hip disease originates in the acetabulum. This is more common in adults than in children, but is not unknown at an early period of life. In children it most frequently begins in the neighbourhood of the triradiate cartilage, and early infects the joint. The abscess which results is commonly intrapelvic at first. It may afterwards work its way upwards, bursting the pelvic fascia and pass into the psoas muscle, pointing below Poupart's ligament, or in the iliac fossa. In other cases it is discharged through rectum or vagina. Occasionally it escapes through the sciatic notch. This form of hip disease is most hopeless, whether it be regarded from the view of prognosis or treatment. It is frequently associated with wide-spread disease in the pelvic bones. Clinically it is almost impossible to separate acetabular from the purely synovial form of hip disease. In many cases this can only be done when we find the symptoms of hip disease associated with the presence of an intrapelvic or psoas abscess.

In other cases the disease begins in the synovial membrane, frequently in the neighbourhood of the ligamentum teres, which is destroyed at an early stage. If abscess results, it most commonly escapes by destroying the capsule posteriorly beneath the gluteus minimus. In other cases it passes forwards between gluteus minimus and rectus femoris, and then between sartorius and tensor fasciæ femoris, pointing just beneath the anterior superior spine, or it may distend and rupture the bursa beneath the psoas.

Of all the bursæ around the hip those most frequently the seat of tubercular infection are the bursæ separating the glutei from the great

trochanter, and from that situation disease may readily spread to the joint.

Clinical observation of many cases in which arthrectomy or excision has been performed in this hospital, appears to me to point out that tubercular arthritis of the knee is most commonly synovial. The synovial membrane is generally found to be the site of the original infection in such operations, and it is, of course, the most probable one in the majority of those cases which recover without operation. For the purely synovial form of disease is much more likely to prove amenable to treatment than is disease of the joint associated with simultaneous disease in the neighbouring bones. But in many cases the arthritis spreads from an adjacent bone. Of these the tibia is the one most frequently affected, but the femur, and in rarer instances the patella, may first be invaded. It sometimes happens in this and other hospitals that a child is admitted with an abscess near a joint, the joint itself being healthy. This abscess is opened, exposed bone found and scraped. The wound heals, and the patient goes home apparently cured. The parents allow him to use the limb, and he returns later with tubercular arthritis of the joint. If an operation be performed, the original disease is found as a cavity in one of the bones with or without a contained sequestrum. The abscess was the manifestation of the disease lurking in the bone. I remember one patient who had an abscess near the outer tuberosity of the left tibia, which was opened and scraped out. The boy was discharged with the wound healed, and no signs of disease in his joint. He returned in two or three months with acute tubercular arthritis of the left knee. This was opened, and found full of pus. Further investigation showed a carious cavity in the outer tuberosity of the tibia containing a small sequestrum, and leading by a minute aperture into the joint. I may mention, in order that you may be certain that this was truly tubercular and not a septic arthritis, that the pulpy granulation tissue so distinctive of tubercular arthritis was present in large amount. It is not common for disease of the knee-joint to have originated in the surrounding bursæ, but I can instance two such cases. In one it began in the pre-patellar, and in a second in the supra-patellar bursa. In all other joints tubercular disease most frequently spreads from the articular ends of adjacent bones.

I have spoken of the diagnosis of ostitis in the upper epiphysis of the femur. It is more easy to distinguish this disease in the other bones around joints if it be seen sufficiently early. In such cases, there will be freedom of movement in the joint with no synovial distension, and at the same time, tenderness elicited by making firm pressure upon the affected bone, with occasionally one especially tender spot. Absence of distension and freedom of movement are, however, only present at an early stage; later, as the ulceration approaches the joint surface, simple inflammation is set up within the joint, causing distension with limitation of movement, obscuring diagnosis, and preparing a suitable resting-place for the tubercle bacillus.

After tubercular arthritis has long run its course, the neighbouring bones become secondarily involved, and in many cases so extensively ulcerated and necrosed, that it is impossible to remove all the disease, except by amputation above the affected joint. In persistent cases of hip disease, we frequently find extensive caries or necrosis in the pelvic bones. These hopeless results which follow upon neglected hip disease afford (to my mind) sufficient reason for excision when once an articular abscess has formed.

It is not common to find a tubercular synovitis affecting several joints simultaneously, or in rapid succession. In this the disease differs from other forms of arthritis, such as rheumatism, pyæmia, osteo-arthritis and syphilitic arthritis. Tubercular ostitis, involving the bones around many joints, is not uncommon. There are at present two instances of this multiple form of ostitis in our wards. One boy has had one leg amputated above the knee for very severe tubercular arthritis in that joint, and has at the present time tubercular ostitis affecting the tarsus and ilium of the other limb, with almost total destruction of one ulna bone. Another child has had amputation performed at the hip joint for intractable hip disease, and now has tubercular ostitis in the other foot, with arthritis of both elbow-joints, spreading in one case from the ulna, and in the other from the lower end of the humerus. In some cases, however, one does see a form of synovitis resembling the tubercular form in every feature, in which many joints are affected simultaneously or in rapid succession. There are at present three children in our wards suffering

from this variety of synovitis. One boy has had tubercular disease in both knees; on the one side it was preceded by an extra-articular abscess such as I have already described. In his case the diagnosis of tubercle is beyond doubt, as one knee has been excised, when the appearances found were typically tubercular. Another child has disease in the right interphalangeal joint of the left great toe, with chronic synovitis in the right knee and ankle. The third child has disease in the metacarpo-phalangeal joint of the right index finger, with synovitis in both knees and in the right ankle. In these cases, before one diagnoses tubercular synovitis, it is necessary to eliminate all other possible forms of chronic synovitis, and this has been clearly done in both the cases I have mentioned. This form of tubercular disease in joints frequently runs a very passive course, with little pain or tenderness, and only slight limitation of movement. It appears strange that tubercle so widely diffused does not speedily end in death from tubercular meningitis, peritonitis, or phthisis, but this does not appear to be a common termination in these cases. As in the joints, so in the general system, the tubercular infection appears to be mild in character.

I shall only speak briefly of the treatment for tubercular arthritis. Too much stress cannot be laid on the necessity of early and complete rest for the affected joint. By its aid we can often completely cure the disease in its earliest stages, and leave the patient with a useful limb. Personally, I have most faith in the treatment by means of weight extension, continued until all pain and tenderness have long subsided. Others advocate different methods of fixing the joint, and it is probably only a question of individual choice in selecting the manner of doing this. It is only after an abscess has formed in the joint, or when grating during movement, rapid increase in swelling with displacement of the bones, or very severe starting pains unrelieved by extension, point to destructive changes beyond the power of unaided nature to cure, that we should operate. But when these signs are present, nothing short of operation can hold out any great hope of ultimate recovery. In such cases there are four possible lines of treatment. Firstly, merely opening and draining abscesses as they form. This is an uncertain method. Dead bone is frequently present,

and it takes a long time before such sequestra become separated and discharged. During that time there is constant danger from hectic fever, lardaceous disease, or in rare instances general tuberculosis supervening. The second line of treatment can only be adopted when the disease is purely synovial—it consists in the removal of all the diseased synovial tissue, and may be expected to cure that form of the disease. After this erasion we occasionally see a fairly movable joint. The third method of treating tubercular arthritis is by excision. In this hospital I have never seen a true excision performed, except in cases of hip disease. In operating on other joints, the surgeons merely remove the diseased bone, carefully leaving that which is healthy. Thus a carious cavity is scraped or excised, and sequestra are removed; in this way the epiphysal cartilage is but little interfered with, and shortening is rare, excepting so much of it as depends upon the mal-nutrition of the bone associated with the disease. Finally, in some inveterate cases, amputation becomes necessary. This is most common after disease in the tarsus; for when once the complicated synovial membranes of the foot become infected, excision is almost hopeless. In such cases, Sime's amputation removes all the disease, and leaves a very useful stump. In the hip joint, also, it is occasionally impossible to arrest the disease. Extension and excision fail; the patient's thigh becomes riddled with sinuses; he falls into a condition of hectic, or lardaceous disease supervenes. Amputation at the hip joint is in such cases fraught with great danger, but it is, at the same time, the only resource left to the surgeon, and it has always seemed to me to be permissible to subject a patient to even great immediate danger when he is suffering from a painful disease almost certain to prove ultimately fatal. It is wonderful to see how such patients regain strength and put on flesh after a successful amputation. Happily, amputation for tubercular arthritis must become rarer the more we study the pathology and treatment of the disease.

Sycosiform Eczema of the Lower Lip.—

R	Styrax	3 1 $\frac{1}{2}$
	Oil of sweet almonds	...	f 3	3 $\frac{3}{4}$	
	Precipitated sulphur	...	gr.	7 $\frac{1}{2}$	

Jour. de Méd. et de Chir. Prat.

CLINICAL LECTURE
ON
**THE TREATMENT OF CANCER BY
INJECTION.**

Delivered at St. Bartholomew's Hospital, Dec. 11, 1895,

By **H. T. BUTLIN, D.C.L., F.R.C.S.,**

Surgeon to the Hospital.

GENTLEMEN,—A new treatment of cancer, if it rests on, I will not say, scientific grounds, but on grounds which may be regarded as reasonable, and if it is placed honestly in the hands of our profession, is sure to excite a certain amount of attention. For, although we are now able to operate for cancer on parts of the body where we formerly never dared to trespass, there remains, and always must remain, a large number of cases of malignant disease, in which no operation would ever be performed by any reasonable surgeon. There are other cases, in which the patient is unwilling to submit to operation, but is quite willing to undergo long and painful treatment. There are many other cases, in which operation has been performed, and in which the disease has recurred beyond the hope of relief by operation.

To-day I propose to give you an account of one of the latest methods of the treatment of cancer, viz., by means of the injection of the toxins of erysipelas. Unfortunately, my personal experience of it, from cases actually treated in the hospital, is very small. I have only treated three patients by this method, and that is due to the fact that only quite recently have we been able to obtain the fluid in sufficient quantity and of sufficient quality in this country for regular use. However, the history of the cases is not without interest, and I propose to give it you, so that you may know what we have in mind with regard to this form of treatment of malignant growths.

M. H., æt. 68, a labourer, was admitted into Colston Ward on April 23rd last year, suffering from large masses of glands on both sides of the neck and in the axilla, and extending beneath the pectoral muscles. I diagnosed the case as one of lymph-adenoma or possibly lympho-sarcoma, and put him on ℥ij liquor arsenicalis three times a day after meals; this dose was rapidly increased

until he took ℥xij three times a day. This produced symptoms of arsenical poisoning, and the glands rapidly subsided. He got so much better that I had hopes of him. The patient thought he was on the high road to cure, and left the hospital very satisfied. In October of the same year (1894) he was again admitted. He had ceased taking arsenic soon after leaving the hospital; the swellings returned and continued to increase, and when he was readmitted they were larger than on his previous visit. He had a huge mass on the left side of the back, which implicated the muscles of the scapula, and apparently affected the bone itself. On the 25th October I injected ℥ij of a solution of erysipelas toxins, which had been prepared in this hospital, the administration of arsenic on this occasion having had no effect in checking the growth. I do not know whether the erysipelas culture was from a bad or a mild case; that is said to have a great effect upon its efficacy, or otherwise. It is possible that in some way the preparation was not properly carried out. At all events, none of the characteristic reaction followed, and I was about to give up the treatment. But it so happened, about this time, that Dr. Coley, of New York, whose name is associated with this method of treatment, was good enough to send me a copy of one of his papers on the subject. I acknowledged it, and told him I hoped to use the treatment here in England. He replied by asking Dr. Buxton, his bacteriologist, to send me a supply of the fluid. I was, of course, exceedingly obliged to him, and began to use it at once. On the 29th October the patient was injected with ℥ij of Dr. Coley's fluid, which consists of the toxins of erysipelas and bacillus prodigiosus, because he thinks that this bacillus may modify and aid in some desirable way the toxins of erysipelas. Within an hour of the injection the patient had a smart rigor, the temperature rose to about 103°, the pulse and respiration quickened, he suffered from headache and general malaise. These symptoms continued for some hours, gradually subsiding, until, on the following day, the patient was practically as well as he had been before the injection. The injections were repeated almost daily, and on nearly every occasion the characteristic reaction ensued. At the end of three weeks the poor fellow was very much exhausted by the treatment, particularly as he was not in very good

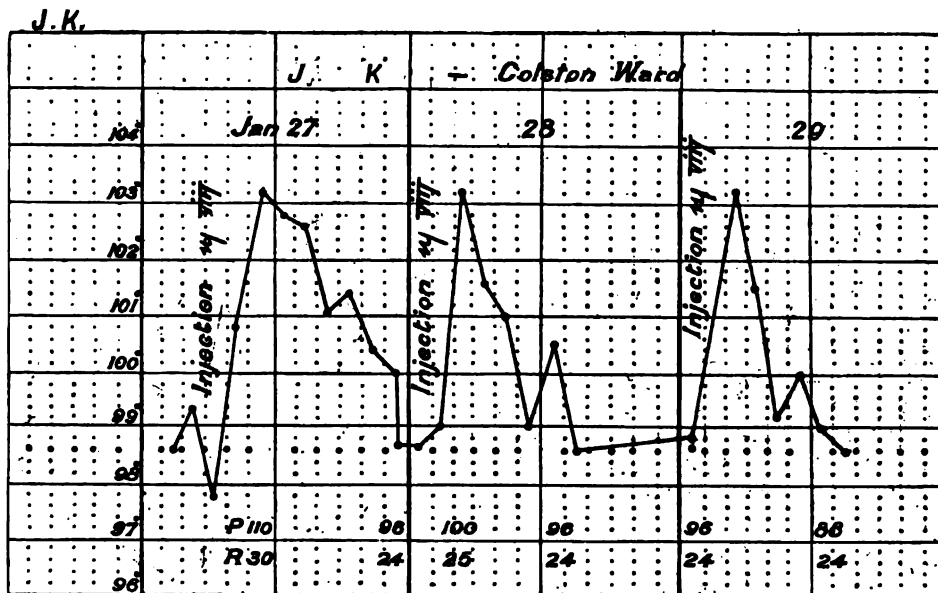
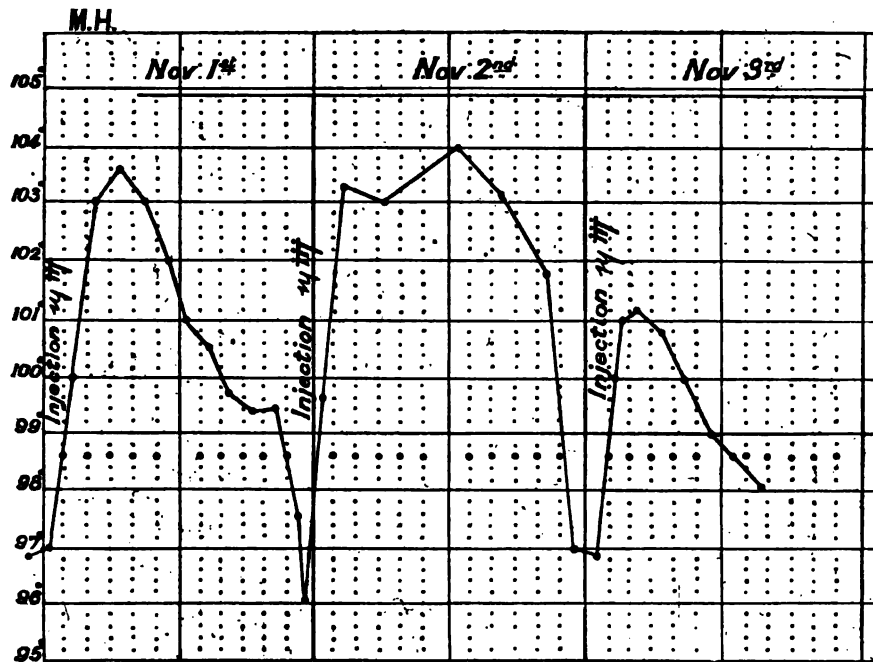
condition when it was commenced. I was therefore obliged to interrupt it and send him down to Swanley for a time to recruit, in the hope that I might be able to repeat the injections. On December 17th he was readmitted in a dying condition. Respiration and the circulation, especially in the upper extremities, were very much impeded by the size and position of the growths; and on December 23rd he died. Of course no injections were made during these six days. At the post-mortem examination large masses of growth were found, not merely in the glands, but in the muscles and other parts; and some of these growths were calcified or ossified (I do not know which, because no microscopical examination was made, on account of the date being so near Christmas). I think you may take it that the disease was probably sarcomatous, perhaps lympho-sarcoma, or some form of round-celled or spindle-celled sarcoma.

My second case is that of J. K., æt. 45, a labourer. He was admitted on January 25th, this year, suffering from a huge mass in the left side of the pelvis and abdomen, a great sarcomatous tumour, which appeared to be connected with the iliac bone. His disease had existed for about five months, and he was very much wasted. On January 26th I commenced to inject him with the fluid which Dr. Coley had sent me, and the characteristic reaction followed—a rigor, high temperature, quickened pulse and respiration, headache, and general malaise. The injections were continued to February 14th, during which time the dose was increased to ℥viii. On February 14th a very unfortunate accident happened—I exhausted my supply of Dr. Coley's fluid—and, in order to keep up the injections (up to that time very little change had been perceived, and I thought it desirable to continue the treatment from day to day) I used some fluid which was prepared in this hospital. The effect was very curious—the man had a rigor, his temperature rose to 103° or 104°, just as it had done before; but, instead of getting better in 24 hours, he remained ill, and his temperature did not fall. After a time he first ejected and then rejected, his food, and he died of asthenia on February 21st. I am not surprised that he did die, because at the post-mortem examination we found not only a huge round- and spindle-celled sarcoma growing

from the ilium and filling up the pelvis on one side and extending into the abdomen, but also secondary deposits in the kidneys, spleen, liver, lungs and heart. So that, I take it, neither Dr. Coley's nor any other fluid would have been likely to produce any good effect upon him. I show you the temperature charts of the two cases.

At the present time there lies in Paget Ward a poor woman, who is suffering from a large tumour, high up on the inside of the left thigh. She says she has had it six years. It grew very slowly indeed, and caused her very little trouble; and partly for this reason, and partly because she did not like to come to the hospital to show a growth in such a position, she has kept it all this time. Now she is actually driven here by the pain she is suffering, and because, in addition to the primary disease, she has enlarged glands in the inguinal region, extending into the pelvis. On September 24th I commenced to inject her with the solution of toxines of erysipelas, which is prepared under the direction of Dr. Ruffer, at the British Institute of Preventive Medicine. The characteristic reaction followed on almost every occasion. We commenced with ℥j, and increased to ℥ij, to ℥iiiss, and finally to ℥iv. Certainly *every* injection has not been followed by rigor, but nearly every one caused a rise of temperature, headache, and malaise. In fact the poor woman has been exceedingly ill, so much so that I was obliged to remit the injection on some days. The degree of disturbance produced varied somewhat, so that the dose had to be varied. Quite lately I have had another supply of Dr. Coley's fluid, but not direct from him. I obtained it from a patient, to whom it had been sent with the hope that he would be injected. We were able to compare the effects produced by the two preparations, and we found no difference in the effect of Dr. Coley's fluid and of that supplied by the British Institute of Preventive Medicine.

Once or twice I have looked at that poor woman, whose case was not one for amputation because the glands also are affected, and wondered whether it was wise to continue the injection treatment. But she has suffered so much pain, and is so anxious to have something done, that it seemed only reasonable to give her so much chance as the new treatment might afford. I confess I kept on the injections with great lack of will, and felt con-



science-stricken at seeing her so ill every day. If I had given this lecture a week ago, I should have said that no good whatever had followed the injections. But when I examined her last Thursday I found considerable changes had taken place. The main tumour does not, to me, seem much improved, but the skin over it has become slightly livid, and there is a sense of semi-fluctuation which was not previously present in the growth. I cannot but think that the tumour is breaking down, and that this may be the effect of the injections. Again, the glands were steadily increasing in size; last Thursday, though they were still fixed, and very large, they were very soft, and there appeared to be a collection of fluid. I therefore asked Mr. Adams to draw this fluid off, and get it examined. He found it difficult to draw off; it seemed to be so thick and grumous.

I therefore cannot say that any of the cases which I have treated, or the cases seen in consultation, which I have advised should be treated by this method, have received any markedly good effect from it. I am wondering what will happen to this poor woman, and I am rather disposed to inject again into the lower part of the tumour.*

Now, it may be very fairly asked why it should have entered into the mind of any man to inject the toxins of erysipelas into a malignant tumour; what have the two things to do with each other? I shall have to answer that this treatment is based on just as good grounds as a large number of other treatments which we adopt for various diseases. It is just as reasonable as the administration of quinine for ague, or of iodide of potassium and mercury for syphilis. I suppose that, in the first place, the specific action of those remedies was discovered by accident. Then, by continual experiment, by again and again repeating the administration of the medicines, they came to be regarded as specifics for those particular diseases.

About the year 1866, I think, Professor Busch, of Bonn, a very distinguished surgeon, noticed in a certain case that a malignant tumour disappeared after the patient had been attacked by erysipelas.

* I am sorry to say that this tumour has steadily grown larger, and only a portion of its surface has sloughed.

The softening of a portion of the glandular mass, which I hoped might be due to fatty degeneration, has been accompanied by increased growth of the solid portion of the mass.—January 10th, 1896.

He looked into the matter, and found that other persons had now and again observed either complete disappearance of a malignant tumour or marked reduction in its size, under similar circumstances. I have myself noticed the same thing, and I can quote a case which occurred in this hospital. I remember a boy who suffered from malignant tumours at the back of the leg; he had an attack of erysipelas, one of the tumours disappeared, and was not reproduced at that point. If you want to know the whole history of this matter you cannot do better than read an excellent article on the subject by Professor Paul Bruns, of Tübingen, published in 1888,* in which are narrated all the cases collected from medical literature in which malignant tumours had been distinctly affected by an attack of erysipelas, and others in which no noticeable effect on the growths had been produced by erysipelas. Bruns had observed one instance himself—a case of melanoma of the breast. He removed the disease; it was examined microscopically, and there was no doubt as to its nature. Almost before the wound had closed, fresh nodules appeared, and some of them ulcerated. Then the patient had a smart attack of erysipelas, which appears to have swept over this tumour, and cleared it away; the nodules disappeared, and the patient got, and remained, quite well. A few years afterwards there had been no recurrence of this melanotic sarcoma. Bruns found that there were accounts of five persons suffering from sarcoma who had been attacked by erysipelas, and three of them had been cured of their tumours. But there were three cases of epithelioma in which the patients had an attack of erysipelas, and the attack did not appear to afford relief in the smallest degree. He also found that in six cases of malignant tumour, in which it was uncertain whether the disease was carcinoma or sarcoma, no improvement whatever was effected by an attack of erysipelas. It occurred to Busch and to Bruns also whether it would not be proper and reasonable to produce an attack of erysipelas in a person suffering from malignant disease which could not be treated by operation; and it is a most interesting point, in connection with the contagion of disease, to study the methods which were adopted to produce erysipelas in persons who previously had not suffered from it. Unless the

* *Beträge z. Klin. Chir.* iii., 443.

patient had an ulcerated tumour, of course it was necessary to make a wound. The discharges from people suffering from erysipelas were placed upon the open wound, but that did not impart it. Then the dressings, all dirty with discharges from erysipelatos patients, were applied, and still the result was negative. Even when Fehleisen's coccus was inoculated it did not always produce erysipelas, though it generally did so. However, Busch did succeed in inoculating five people suffering from malignant disease, viz., two from carcinoma, two from sarcoma, and the fifth uncertain. The two patients suffering from carcinoma were improved somewhat; but those suffering from sarcoma were not in the least improved.

A surgeon named Janicke inoculated a patient with erysipelas, and the erysipelas produced a very remarkable effect upon the tumour, which underwent rapid degeneration and absorption. But it produced a worse effect upon the patient, for, four days after the inoculation, he died. I had it in my mind some time ago to try the effect of inoculating some patients with erysipelas, and I would have done so had they been willing to submit to it, because it seemed to me the inoculation might give some kind of chance to people who otherwise would have none, either of relief or cure. But I did not do it, because when I had the proper material to inoculate, I could not find a subject suitable for the treatment. Some two or three years ago Dr. Coley, of New York, studying the later developments of bacteriology, and the separation of toxins from pathogenic micro-organisms, thought—and about the same time the same idea occurred to two German surgeons, Emmerich and Scholl*—that the toxins might produce the same effect as an actual attack of erysipelas. Coley, therefore, had the toxins separated from the erysipelas germs, and used them for injection. He has published papers on the subject from time to time†; other people have also employed his fluid, and have published their results. Coley's last paper contains the results of treatment in 38 cases of sarcoma and 19 cases of carcinoma, and these results agree in a very curious way with the results which were formerly given by Bruns, taken from medical literature. Of

the 38 cases of sarcoma, nine appear to have been completely cured by the injections; and as the disease in most, if not all, of these cases was proved by microscopical examination to have been malignant and sarcomatous, we may take it that these patients were enormously relieved, if not cured. In some the disease was of huge size. The best effect seemed to be produced in cases of mixed-celled sarcomata or in cases of pure spindle-celled sarcomata. Of the 19 cases of carcinoma treated by Coley, none were cured by the injection, and I do not think there is anything to show that any of them were relieved. The conclusion is that in a certain number of cases of sarcomata, erysipelas, or the toxins of erysipelas, does, or may, produce a very decided effect, whereas in carcinoma both Bruns and Coley agree that up to the present time erysipelas appears to produce no sufficient effect to make it worth while to continue the injections. I have carefully looked up the literature of the subject, which is of about two years' duration. Emmerich and Scholl make a slight difference; they separate the toxins from the erysipelas micrococci, and before passing them directly into the human subject, pass them through an animal. This does not result in any diminution of the effect of the toxins on the tumours, but the feverish reaction is almost abolished. Coley appears to have been doing the same thing during the last few months, but I do not think he has tried it sufficiently long to be sure of the results.

What is the effect produced upon, say, sarcoma? It appears to be of two kinds—viz., in the first place, the cellular elements of the tumour undergo fatty degeneration, the tiny molecules are taken up and absorbed, and the tumour shrivels away, but does not break down. That is why I had it in my mind that the state of the tumour in the poor woman whose case I last related may be a step towards improvement. At all events, it may be an *intent* to cure on the part of this fluid. It may not succeed, perhaps, partly on account of the large size of the tumour. Another effect which has been produced on some cases has been that the whole or large parts of the tumour have sloughed away. That seems to me a very dangerous end for a great tumour which perhaps involves large vessels and nerves. I should not be very comfortable if I thought the whole of that

* Centralblatt f. Chir. 1895, p. 764.

† Transactions American Surg. Assoc., 1894. American Med. Record, 1895, Jan. 19th and May 18th.

mass in the woman's thigh would slough away; it would leave a most horrible condition. If the result is to be sloughing of very large tumours without the certainty of cure, it would be better to suspend injections before that happens.

Then arises the important question: How are these effects produced? Are they the result of something specific in the toxins of erysipelas, or of the general fever which is produced by the injection? It is not a continued fever, but a very smart attack which occurs every day. There have been some differences of opinion on that point, but Coley in New York, and Emmerich and Scholl in Germany, are agreed in believing the effects to be due, not to the fever, but to some specific action upon the tumour. I cannot tell you all the grounds upon which this opinion is based.

As to injection treatments generally, you know that various substances have been injected for several years past to cure a number of diseases. I may say it is the fashion of the day to inject, and sometimes patients who are called back to life from some very serious condition, such as syncope or collapse after operation, by the injection of strychnia and the like, become very ill and actually die of the effects of the injection. For the cure of cancer various injections have been used. For instance, injections of anylin were recommended in 1891 by a German named v. Mosetig-Moorhof.* His idea was to produce anatomical and physiological effects. He found that anylin dyes which are used for colouring specimens made their way into the nuclei and *fixed* them, and he thought that the same material injected into malignant disease in living persons might *fix* the nuclei of the cells in a similar manner and destroy their activity. Some three years ago I removed a malignant tumour of the tongue of a patient, who afterwards had the glands affected. The anylin was injected, but he derived no benefit from it; and I cannot find record of any person having been cured by the treatment.

The injection of *cancroin*, which is much more ambitious, was recommended some years ago by Dr. Adamkiewicz.† He laid down the law that carcinoma is a parasitic disease, and that the curious forms of cells which are observed in

epithelioma are parasitic. Then he extracted from cancer a material which he injected into animals, and made them horribly ill. He called this material *cancroin*; and, as it was not easy to obtain a sufficient quantity of malignant disease to procure as much *cancroin* as he wanted, he extracted from dead bodies a material which he considered equivalent to *cancroin*, and which produced much the same effect. In making out the chemistry of this substance, he found it equivalent to tri-methyl-vinyl-ammonium-oxy-hydrate, which much resembles, if, indeed, it is not identical with *neurine*.

This material, when injected into the disease or into its neighbourhood, produced no effect upon the normal epithelium, but a very sensible effect upon the cancer cells; they became shrivelled, and he hoped he would be able to cure cancer by these injections. In his book, which I have here, he relates 20 or 30 cases of cancer of different parts of the body. The end of it is that, although the patients seemed to find the injections very beneficial, not one of them was cured. Therefore I am afraid we cannot accept the theory of Adamkiewicz and the effect of the injection of *cancroin* on carcinoma for the present. The basis of that treatment you may put down as serum therapy, such as is used against diphtheria, tetanus, and the like.

The injection treatment of cancer is much older than this—for all I know, the ancients may have injected cancers—for Dr. Broadbent (now Sir William) published, in 1866, a little book on "Cancer: a New Method of Treatment by Injection,"* in which he recommended the injection of acetic acid. Until a few days ago I had no notion that that suggestion came from Sir William Broadbent, although I can remember some trial of it in the hospital at the time I was a student. Sir William's theory was precisely similar to that of Moorhof. He chose acetic acid because it would not coagulate albumen, and because it might be injected into the vessels without producing serious effects. He found that in microscopic specimens it made its way into the cells and modified them, and he thought this might happen in the living subject. The book I have mentioned relates six cases treated by this method, but I

* Centralblatt f. Chir. 1891, pp. 391—394.

† "Untersuch ueber den Krebs," Wien, 1893.

* London, 1866.

cannot find they were very sensibly benefited; certainly none of them were cured.

To go back to the treatment I have been employing: it is based on very much better grounds than most of those which have been put forward for the cure of cancer; it is the most scientific of them. Although my own experience of this material at the present time is not very encouraging, yet I shall continue to use it for some months, perhaps for a year or more, to come. I hope by and by we shall see definite results, and that experiment may enable us to decide in what cases it may be advantageously employed, and whether the same results are obtained when the toxins are passed through the body of an animal before they are injected into human beings. To people who ask whether I recommend the fluid for carcinoma, I reply, unhesitatingly, "No," because up to the present time it has not been shown to affect those growths, and particularly because the fever produced by the inoculation of erysipelas toxins is not justifiable unless there is a decided prospect of improvement or of cure. I shall use it for sarcoma, and in cases where the nature of the growth is doubtful, and hope, on a future occasion, to relate to you the results.

The fluid can now be obtained from the British Institute of Preventive Medicine,* where it is prepared by Dr. Armand Ruffer with the greatest care, not only to obtain the toxins of the erysipelas coccus and of bacillus prodigiosus, but also to ensure its freedom from every impurity. I think it is well to begin with an injection of not more than ℥j, in order to test both the strength of the fluid, and the susceptibility of the patient. The injections should be made in or in the neighbourhood of the tumour, and should, if possible, be repeated every day. Dr. Coley is of opinion that daily injections for a fortnight will produce a sufficient effect to show whether the treatment is likely to be useful in the individual case.

* Temporary Office, 101, Great Russell Street, W.C.

For Gastric Disturbances in Anæmia.—

R Ferri sulph.
Papain āā gram. 3'6
Ext. cann. Ind.
Ext. nuc. vom. āā " '45
Ext. rhei " '9

M. ft. pil. Divide into 60. Sig.: Two pills after each meal.
Corresp. fur Schw. Aerzte.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London
Clinical Society, North-West London Hospital,
January 15th, 1896.

WILLIAM BOULTING, Esq., in the Chair.

Torticollis Simulating Writer's Cramp.

Dr. CAGNEY exhibited a man, æt. 70, the subject of torticollis. He said the case was remarkable in itself, and was of peculiar interest on account of the element of confusion which had crept into similar cases. He owed to the Chairman the opportunity of bringing it before the Society. The condition was complicated, and rendered to a certain extent obscure by an affection of the same nature involving the right upper extremity. The patient's previous history was uneventful up to the date when the condition distinctly commenced; he had had no specific disease, and he (Dr. Cagney) did not think there was anything in his habits bearing upon the matter. When the patient was 25 years of age he strained his back by carrying a heavy weight, and felt subsequent pain, but that was evidently located in the sacro-lumbar aponeurosis, and had no connection with the present illness. Fourteen years ago he had a fall on the shoulder, and a year later was unable to write. The difficulty consisted in a tendency of the pen to dig through the paper, and up to six months ago no other symptom could be detected, so that for the patient to believe he had writer's cramp was only natural, particularly as he was a commercial man. Six months ago he was taken with cramp in the neck, which caused the head to turn slowly to the right; and in order to keep it still he had to vigorously restrain it with his hand. But the jerky movement to the right having once commenced he could not check it until it had run its course—i.e., until the chin pointed directly over the apex of the shoulder. During the spasm the patient complained of a sensation which he likened to the presence of a cat's paw on the left shoulder, and a feeling on the left side of the face as if he were brushed with a feather. In the ordinary way, sensory troubles were not conspicuous in this disease. The spasm was

rhythmical, varying in rate, and did not occur in sleep. When the patient sat in a deep-seated chair with a long arm, with the back of the chair just supporting the neck, he was quite at rest. There was no facial distortion, the tongue protruded straight, pupils reacted to accommodation and light; the corneæ were clouded, and showed a complete arcus senilis; the urine contained no albumen nor any sugar. The point of most interest was the affection of the arm 14 years ago, which might have led an observer, who was not very careful, to diagnose the affection as writer's cramp; but the mystery was solved 13½ years after the fall. Still, at that time, even when the spasm commenced in the right arm, if the patient's hand had been rested on the observer's, both being at arm's length, a movement of the patient's arm across the chest—an adduction, and pronation of the forearm—comparable to the movement which caused the final jerk of the implicated muscles, would have been noticed. These movements were demonstrated by Dr. Cagney. The localisation of the spasms was such as is often met with—the implicated muscles seemed to be the trapezius and sterno-mastoid on the left, and the recti and oblique muscles of the right side. It was not rare for one upper limb to be engaged, as was the right arm in this case, but a commencement of the spasm there was quite unusual. Perhaps, however, such a commencement was more common than was supposed; and these cases were mistaken at the outset for cases of writer's cramp. The long interval of 13½ years which had elapsed here before the typical spasm of the neck set in showed plainly that the error once made might remain long uncorrected. Accurate observation, however, would make such an error at any time impossible.

Mr. MAYO COLLIER, in expressing his gratitude for the opportunity of seeing the case, said that, though we knew something of the treatment of torticollis, we were still in the dark as to its pathology. As to treatment it was remarkable that some cases got completely well by merely stretching; in others section of the nerve was efficacious, though in others neither nerve section nor removing an inch of the nerve abated the disease. He described a case of torticollis in a young woman of 22, in whom the spasm was so severe that it raised the head from the pillow.

He decided not to cut out the nerve until he had tried tentative measures, and, therefore, he cut down to the nerve, and placed round it a fine silver wire, which, if necessity arose to excise the nerve, would lead him down to the part. But as soon as he had placed the wire around the spinal accessory nerve the spasms ceased, and the patient had had no recurrence, and was as well as if she had not been affected. Possibly in twisting the wire round he came down to the axis cylinder, and that the nerve force which previously had caused the spasms escaped by this channel into the tissues.

Dr. GUTHRIE said it was difficult to account for the condition, and he did not see how any one lesion would explain it. It was most probably of the nature of a habit spasm. In writing, particularly with illiterate people, the head was always inclined towards the left; that is, the sterno-mastoid on the right was continually in action, and in time became powerless, owing to constant use; then the left sterno-mastoid would take on the action of the right, resulting in the head turning by spasmodic movements.

Dr. CAMPBELL also commented on the case.

Congenital Absence of Abdominal Muscles.

Dr. GUTHRIE showed an infant, 9 weeks old, in which the abdominal muscles, external to the recti, were apparently absent. It was born at full time, and there appeared to have been no difficulty in labour. "Something wrong" about the abdomen was noticed at birth, but the mother could not explain what. The child had wasted since birth, and continued to do so. The abdomen was particularly flaccid and large, inelastic, and the skin much wrinkled in longitudinal folds. There was no trace of an ordinary umbilicus. The cord was said to have been a long time coming away, and some hæmorrhage followed. The recti muscles could be distinctly felt between the fingers, but beyond that there was no trace of muscle; the abdomen sagged from side to side, and the intestines could be seen moving on either side of the recti. There was some prominence in the lower part of the abdomen, which he had thought to be a congenital myxomatous tumour, but it had turned out to be a distended bladder. Large quantities

of urine were passed. From the wrinkled state of the abdomen he thought it quite possible that the child had had ascites *in utero*, which had subsided, and that probably the distension *in utero* caused atrophy or want of development of the abdominal muscles referred to. He had been unable to find record of a similar case.

Mr. JACKSON CLARKE said the very rare case before them served to illustrate the importance of the broad muscles of the abdomen in retaining the abdominal viscera. In that child there were practically two large lateral congenital herniæ, and it was easy to see the necessity, in incising the abdomen down to the peritoneum, for having muscle in front of the scar representing the opening.

Dr. CAMPBELL said it occurred to him that the diaphragm might be absent, as the ribs were elevated considerably at each inspiration, and he could not detect any increase in abdominal tension. He did not think ascites would have caused complete atrophy of one set of muscles without affecting another set.

Mr. MAYO COLLIER said he had peculiar ideas about the importance of the muscles of the abdomen and chest and the diaphragm, as agents in carrying on the circulation, and he thought it was too much to say that in the case before them there was complete absence of the abdominal muscles. They were not warranted in saying more than that those muscles were not in their normal state, but were extremely attenuated, or badly represented. If such muscles were absent, he maintained that the child would inevitably die. He thought pressure from ascites *in utero* probable; or possibly indigestion later led to atrophy of the muscles and protrusion forwards of the abdominal walls, such as is met with antecedent to hernia, or after fevers, or long-existing diseases. The case was one which should not readily fade from their memories.

Dr. GUTHRIE said he believed the diaphragm existed, and as to whether there was absence or merely atrophy of the abdominal muscles time would show.

Result of Bassini's Operation for Radical Cure of Hernia.

Mr. JACKSON CLARKE presented a man, æt. about 38, on whom he had successfully performed

Bassini's operation recently. The man came to him a few weeks ago with strangulated hernia, and all that was now left was a linear scar, and some slight thickening of the tissues, which would pass away in course of time. The practitioner who sent him the case had tried gentle taxis, but without avail. Taxis was, of course, a dangerous expedient if not performed very gently. The operation of Bassini was not altogether a new proceeding. It required the free opening up of the inguinal canal, and the external oblique should be opened two inches above Poupart's ligament, so that the line of suture in the external oblique shall be away from the deeper sutures. Then the cord and sac should be defined and separated, and the sac twisted. The twisting of the sac drew so much on the neighbouring peritoneum that it had been known to cure a hernia on the opposite side. The sac in this case was twisted tight with strong silk and cut off. But the distinguishing point in Bassini's operation was the bringing down of the viscera, the internal oblique and the transversalis muscles, and stitching them to a point outside the internal abdominal ring to Poupart's ligament, so that the whole of the weak part became covered with muscle. The best protection was by bringing down the muscular tissue, which was effected by six stitches, the outermost stitch being outside the internal abdominal ring. The cord was put down in its place, the flap turned up, and the internal pillar and ring finally stitched, so that the cord now, instead of being beneath the border of the internal oblique and transversalis, was well in front of them. The operation was now largely practised, and he (Mr. Clarke) thought it the best we have for the radical treatment of hernia. The patient was in bed a fortnight, the dressing was changed on the ninth day, and the wound soundly healed. He was told to keep quiet for a time, and had since continued his work. If the hernia had been merely reduced under chloroform he would have had to wear a truss, there would have been danger of the hernia becoming strangulated again, and he could not have followed his laborious occupation.

Cerebro-Spinal Meningitis.

Dr. CAMERON showed a young lady, æt. 19, lately the subject of cerebro-spinal meningitis.

He had the patient under observation for four months, during which he saw her once, twice, or thrice a day, taking notes of all his observations, but did not propose to weary the Society with all the details. The patient had always been healthy, and the only sign to the contrary was a little exophthalmic goitre, which persisted. She applied for advice on March 15th, complaining of severe headache, chiefly occipital. A searching examination of the body did not put him on the cause of the headache; pressure upon the head downwards elicited no pain, neither did deep pressure over the cervical region. Thinking the headache might be due to the little dyspepsia and constipation which she had, he tried to cure that, but in three days she returned complaining that the headache was worse than ever. On March 19th she was unable to get out of bed; she was unable to sleep, and had had a very serious night. She had felt giddy and sick, and had pains in the back. He (Dr. Cameron) then thought of cerebral tumour, but examination of the eyes gave no evidence of such, nor of optic neuritis. A week after that other symptoms had developed; she had extreme pain down the whole cervical region, as far as the angles of the shoulders; the head was retracted, and rotated towards the right, so that the chin pointed over the shoulder. The muscles at the back of the neck were extremely rigid, and the grasp of both hands very feeble; she also complained of "pins and needles" and numbness. The legs also were very helpless, rigid and extended, and she complained of great pains in the limbs, with numbness, but pressure on them did not increase the pain. At this date the eyes showed the following symptoms:—ptosis, external strabismus, pupils widely dilated, optic neuritis, photophobia. The condition got gradually worse for another week, at the end of which time she lost control over the bladder and rectum. She could not feel the prick of a pin, nor severe pinching on the legs; she lost the sense of smell, and taste was much impaired, but hearing was preserved throughout. About May 8th, the symptoms began to clear up somewhat; the ptosis had ceased, and the strabismus was disappearing. The rigidity at the back of the neck was not so marked, and the head could be slightly rotated towards the middle line without great pain. She could feel very slightly in the tips of the fingers,

but not above, nor in the legs. Towards the end of May the symptoms had gradually abated, but left the patient almost blind. The pupils were widely dilated. Matters improved up to July 15th, when Dr. Campbell kindly saw her, and confirmed the diagnosis.

The lady was a cashier in a large London establishment. The box-office in which she sat was very small, and in order to enter it she had to stoop and place herself in awkward postures during which she occasionally struck her head on the desk, on one occasion being much stunned. The headache seemed to date its development from that blow. She had recovered in every respect except in her eyes, and it was generally agreed they were in a bad state. He had never detected her temperature above 100°, but the pulse reached 140, and was at about 120 for eight days. He would like to hear the views of members as to the part played in the disease by the striking of the head, and how far it was considered the employer was liable for the sequelæ.

Dr. CAGNEY said it would be practically impossible to question the diagnosis on the facts so carefully noted and recorded; but no systematic tests of the motor paralysis had apparently been taken. This he recommended. The absence of knee-jerks was curious, and needed an explanation. He did not believe anybody was so delicately constituted that the movements to get into the cashier's box would cause the diffuse injury sustained by the patient. Nothing was more remarkable than the habitual resistance of the spinal thecæ to the spread of inflammation from the cranium. He had heard Sir William Broadbent remark that severe cases of cerebro-spinal meningitis had become more common lately, in his experience, than they had been. Dr. Cagney believed that such figured among the sequelæ of influenza. When that was so they gave hope of recovery. Such cases were, of course, distinct from epidemic cerebro-spinal meningitis; but those who believed, as he did, that influenza was the product of an organism resembling the plasmodium of malaria, and bore in mind that epidemic cerebro-spinal meningitis was a disease in which a probably similar organism selected the membranes for attack, might see in influenza an agency which tended to impair the natural resistance of which he had spoken above.

Dr. CAMPBELL thought there must have been some myelitis, but that was not mentioned. He believed the blow on the head was largely answerable for her present condition; if so, the employer should be sued for solid compensation, as she was ruined for life.

Mr. JACKSON CLARKE, while agreeing that the illness was possibly due to the injury, held it was impossible to prove it, and he did not recommend going to law on the matter, in support of which he quoted cases of a similar nature.

Dr. GUTHRIE expressed agreement with the preceding speaker; also with the view of Dr. CAGNEY, that the disease might be a nervous sequel of influenza.

Dr. ALEXANDER MORISON thought it possible that the case was hæmorrhagic, though it may not have been so originally, and the evidence of antecedent exophthalmic goitre showing notable and vascular derangement he held to support his view.

Dr. R. P. LONG said no mention had been made of a cephalic cry or tache cerebrale; but the patient seemed decidedly hysterical. He strongly advised Dr. CAMERON to abstain from going to law on the matter.

After some further discussion Dr. CAMERON said he had never detected the cry referred to. The pain was sometimes so extreme that he had to keep the patient under opiates. She improved immensely under salol, which created a keen appetite; the dose was gr. x four times a day.

Mitral Obstruction.

Dr. CAMPBELL exhibited a heart which exemplified mitral obstruction. There were two well-known kinds of orifice, the button-hole and the funnel-shaped, and he might modestly claim to have discovered that each of these had a meaning. The quantity of fluid which escaped through a vessel was not entirely determined by the size of it. In the case of a round hole, a section of the jet was not the same as a section of the orifice, but was as six to ten, which was the coefficient of the discharge. But in a slit, the coefficient was increased to $\frac{8}{100}$; therefore, for the same area, a slit would permit of quicker escape than a circular aperture. This, he believed, afforded a fresh example of nature making the best of a bad job in obstruction.

Granular Kidneys in a Girl, æt. 18.

Dr. CAMPBELL showed the granular kidneys of a girl æt. 18, who, the night before her death, was working behind a bar. She complained of no previous illness, but was suddenly seized with illness and died, presumably from uremia. As to the pathology of granular kidney, nothing was known.

Dr. GUTHRIE said Dr. Barlow had described a case of granular kidneys in a child æt. 5. The symptoms were gradual wasting; there was pigmentation of the skin, no dropsy at any time, convulsions, and finally death. He himself had had a case in a girl, æt. 7, who came with severe headache and vomiting. There was a trace of albumen at one time in the urine, when the latter was plentiful, while at another time, with a small quantity of fluid would be a large proportion of blood, albumen, and casts. She had convulsions, was slightly paralysed on one side, and after lingering three weeks she died. Post-mortem he found three large hæmorrhages in the right hemisphere of the brain, the largest the size of a Tangerine orange, the smallest as large as a nut.

Ulcer of the Lip.

Mr. MAYO COLLIER presented a man, æt. 30, with ulcer of the lip. He said he brought the case to illustrate the difficulty which arose in differentiating syphilis and epithelioma. Ten days ago, the patient came with an angry raised tumour on the side of the cheek, which had been present for some time, and upon which no treatment had had any effect. A hard gland could be felt in the submaxillary region, both ulcer and gland being perfectly painless. His (Mr. Collier's) first thought was syphilis, while epithelioma also occurred to him. He had scars on the head, which were explained by saying he had an accident, and the wound had rapidly healed; but there was no evidence of syphilis. An examination of the body yielded nothing, and his wife's history was free. When his nose was examined, however, an opinion of the disease could be at once formed, as the greater part of his bony septum was absent, and that was pathognomonic of syphilis. Then the epiglottis was gone, and yet the patient knew of nothing of the kind being amiss. He was put on mercury and iodide of potassium, the ulcer was healed, and

the gland subsiding; therefore the diagnosis was confirmed.

Congenital Ichthyosis.

Mr. HOSFORD (for Dr. Stowers) showed and described a case of congenital ichthyosis, and also microscopic specimens derived from a malignant tumour of the back in a patient exhibited at the last meeting of the Society.

THERAPEUTICAL NOTES.

Dermatol as a Hæmostatic.—Dr. Hecht has applied this material in hæmorrhage with the best results. Originally he used it in bleeding from the conjunctiva after removal of a pedicellated polypus from the eyelid. If the bleeding is severe the dermatol must be applied with pressure on a piece of cotton-wool. In a case of fissure of the tongue and one of tooth extraction, the means proved successful after simple plugging and perchloride of iron had both failed.

Therap. Monatsch.

Whooping Cough.—Otto Fiertz has treated 75 cases with bromoform with very good results. Large doses are required and well borne—for children under 10 years he gives every eight hours (or, if rest is much broken, every six hours) a dose of two drops more than the child's age in years: a child of three, *e.g.*, gets $3 + 2 = 5$ drops each dose. If in a week no improvement is shown, the dose may be increased by one drop. With these doses the author considers the medicament a specific. It must be given in sweetened water, care being taken that the whole dose, whether dissolved or not, is swallowed.—*Corresp. Blat.*

Warts.—

R Trichloracetic acid ... 9 parts
Alcohol ... 1 part

Sig.: To be smeared on once a day.

Or Acid salicyl
Acid lactic ... 2 parts
Collodion ... 4 "

Sig.: To be painted on twice a day.

Deutsch. Med. Woch.

REVIEWS.

The Methodical Examination of the Eye.
Being Part I. of a Guide to the Practice of Ophthalmology for Students and Practitioners. By WILLIAM LANG. (Longmans Green & Co.)

Price 3s. 6d.

To those who know Mr. Lang his name alone will be sufficient guarantee that what he writes will be written well and carefully. This little book of 96 pages is essentially one for beginners. It gives exhaustively step by step the manipulations necessary for determining all the factors essential to a complete diagnosis. Under each heading is given a list of requisite appliances, and how they are to be used. For students it will prove invaluable, but we hope its study by qualified men will become less and less necessary by reason of their previous familiarity with its contents. It is *not* a book on diseases of the eye, but a complete guide to finding disease and its exact locality.

Malaria and its Consequences. By ROBERT LINDSAY, A.M., M.B. (Lewis.)

Price 4s.

An essay on malaria, in which the bacillus malarie is not even mentioned, is certainly very like "Hamlet" without the Prince of Denmark, and certainly this essay seems to us about as useful as the play would be. It is a laborious attempt to make carbonic acid responsible for all the pathology and symptoms of malaria, and we need hardly add that the result is, to say the very least of it, unconvincing.

NOTICE.—Lewis' (136, Gower Street, W.C.) Nursing Charts are excellent little charts for nurses to record observations upon: the hours of the day are ranged in a vertical line, each standing at the left of a horizontal line for temperature, pulse, respiration, bowels, urine, sleep, stimulants, medicine, etc. Their price, 20 for one shilling, or 25s. per 1,000, seems to be moderate enough for anything.

THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 5, 1896.

A CASE OF ACROMEGALY.

By W. J. HADLEY, M.D.,

Assistant-Physician to the London Hospital.

I AM indebted for this case to Mr. Waren Tay, who kindly sent it from Moorfields, where the man had sought treatment for his failing eyesight.

This disease is rather more common in females, but occurs almost as commonly in males.

First, it will be well to give you a brief account of the signs and symptoms of this particular case, and then we shall be able to compare them with those seen in other similar cases.

The man, who is aged 34, first noticed that his hands, feet, and head became much enlarged. This enlargement has been gradually manifested during the last two and a half, or at the most, three years. It has caused him but little inconvenience in his employment as a porter, and he felt perfectly well in himself. It was not until 12 months ago that his eyesight began to fail, and this failure gradually increasing, he applied at Moorfields, where the nature of the case was recognized.

The family history, briefly, is as follows:—His father and mother died at the respective ages of 65 and 70, and had always been healthy people. He has had, in all, eleven brothers, nine of whom died while quite young, and the remaining two are still living and healthy. Personally, but for his present complaint and an occasional bronchial catarrh, he has enjoyed perfect health. He is a single man, and he denies having had syphilis. His employment (as a porter) subjects him to very heavy labour; and he states that, throughout his whole life, he has been a most abstemious man.

Now we may pass to the analysis of his symptoms:—The nature of the enlargement, in the case of the head, is seen to be almost confined to the bones of the face. The malars are enlarged and prominent, the frontal arches protuberant, but the chief enlargement is shown by the inferior maxilla, which is so much increased as to cause it to underhang the upper jaw, and to bring the lower

in front of the upper teeth. In the upper extremities the increase is almost limited to the hands. It will be noticed that they are not lengthened, but that the breadth and thickness of each bone is markedly increased. Almost the same features may be noted in the changes which have taken place in the feet. But here the enlargement has not remained quite limited (though almost) to the foot, for there is also some deformity of both tibiae.

Turning now to the soft parts, we may notice that, in this case, the eyelids and ears are not greatly altered in size. The lips and tongue, however, are much enlarged, especially the tongue and the lower lip, both of which are enormous, and cause considerable alteration and difficulty of speech and deglutition. The nose is also increased in all its dimensions, whilst the soft palate and fauces are somewhat thickened. The marked increase of the facial bones, in comparison with those of the cranial vault, is shown by measurement. The circumference of the head, taken horizontally just above the ears, is 24 in., whilst that taken vertically under the jaw is 27 in.

When we come to examine his eyes, to ascertain the reason for his defective vision, we find that this defect is not due to any error of refraction, nor to any opacities in lens or cornea. With the ophthalmoscope, however, we find atrophy of both discs. It is most marked in the right fundus, the sight of which eye is the worse, and it seems that the inner half of each disc is rather whiter than the outer. Further, on testing with the perimeter, his fields of vision are seen to be much and characteristically narrowed. He is unable to see anything *outside* a central horizontal line with either eye. The temporal field of vision is quite lost. Moreover, the extent of vision is lessened on the inner or nasal side, but to a much less degree, this loss being much more marked with the right than with the left eye.

These, with occasional, but not severe headache, a little dyspnoea on exertion (probably due to his previous bronchitis), the difficulty of speaking and working, consequent upon his deformity, and the

kyphosis of the spine in the cervical and upper dorsal regions, are the signs and symptoms which he presents. His general health is good, and his functions well performed. He, however, occasionally suffers from insomnia.

Let us now compare this with other recorded cases of the disease.

The enlargement of the various tissues in this case are extremely typical, and they alone are sufficient to justify the diagnosis.

The headache and loss of sight are also quite as seen in other cases of this disease.

There are other signs, absent in this case, which are sometimes observed in acromegaly. Thus, in women, changes in menstruation are usually observed. *Hearing* is often partly or wholly lost, and there may be rhythmical tinnitus. The smell may be altered or annulled, or there may be general or mental lassitude and irritability. In some few cases there has been true diabetes, or intermittent attacks of glycosuria. Let us now investigate these various changes and deformities, in order to ascertain their true nature.

The bony enlargements are found to be due to increased size of the bony sinuses and a greater general porosity. When the nose, eyelids, and ears are the seat of change, the enlargement is due to a hypertrophy of their cartilages; by far the greatest amount of overgrowth having taken place in this tissue.

In the case of the lips, tongue, palate, and fauces, the increase in size seems due to a simple hypertrophy of all the tissues composing them, no one tissue being more affected than another. The enlargement of the neck, though in some few cases it may be due in part to a more or less considerable enlargement of the thyroid, is far more usually due to a simple hypertrophy of all its tissues. The general kyphosis of the cervical and upper dorsal spine is accounted for by the great increase in weight of the head which it supports, and also to the fact that, in nearly all cases, a similar increased porosity to that observed in the bones of the face has occurred in the vertebræ.

In considering the pathology of this affection, it would seem that such a widespread, and, for the most part, perfectly symmetrical disease, must have, for its ultimate cause, some central change, some alteration in the nervous or blood-vascular supply of nutrition to the tissues affected. Its

resemblances, in some ways, to myxœdema, led to careful examination of the thyroid, and though in a few cases this gland was found altered, sometimes being atrophied and sometimes hypertrophied, in the majority it was found normal. In seven cases, collected by Dr. Marie, in which a post-mortem was obtained, the changes were found constantly, that is, in every case, in the pituitary body, whilst other parts were not invariably the seat of disease. Briefly, the changes noted in the above-mentioned cases were these:—

1. Increase of the pituitary body in all cases, due to a simple hypertrophy.
2. Hypertrophy of the ganglia and nerves of the sympathetic in some.
3. Persistence of the thymus gland in a few cases.
4. Alterations in thyroid (as mentioned above).
5. Cardio-vascular hypertrophy.

I have drawn your attention to the fact that there is sometimes a difficulty in diagnosing cases of acromegaly from those of myxœdema. Now there are at least six conditions under which somewhat similar enlargements and deformities may occur; and it will be well for us to consider the differential diagnosis between them. They are:— 1. myxœdema, 2. acromegaly, 3. *ostitis deformans* (Paget), 4. *lontiasis ossium* (Virchow), 5. simple giantism, and 6. hypertrophic pulmonary osteoarthropathy.

The hypertrophic enlargement accompanying pulmonary diseases will only give rise to difficulty at a casual glance; for the presence of some well-marked pulmonary trouble, and the want of the typical and extreme enlargement of the bones of the face, will serve to readily distinguish it from acromegaly.

Similarly, in simple giantism, the overgrowth is general, it includes increase in length, as well as lateral enlargement. Again, in some cases, giantism may only affect one or two fingers or toes. Perhaps, however, the enlargement in acromegaly being only lateral, and being, as it usually is, of the hands and feet only, as far as the extremities are concerned, sharply limited by wrists and ankles, serves most easily to distinguish it from giantism.

In the case of *lontiasis ossium* the enlargement is almost, if not entirely, limited to the bones of the face.

In well-marked cases of the three remaining for distinction (myxœdema, *ostitis deformans*, and acromegaly), the face is enlarged in all, but characteristically so in each. The face of myxœdema is rounded and moon-shaped, that of *ostitis deformans* egg-shaped, with the larger end upwards. This is accounted for by the fact that most, if not all, of the changes in this disease take place in the bones of the cranial vault. On the other hand, the face in the case of acromegaly is egg-shaped, with the larger end downwards, by reason that the greatest amount of hypertrophy is always in the lower jaw. Moreover, in none of the other five, and in acromegaly alone, are found the typical eye-changes and limitation of field of vision so characteristic of the affection. It is true it has not been remarked in every case of the disease, but it is possible that there may have been but slight diminution of the field of vision; so slight as to give rise to no complaint of defective eyesight on the part of the patient, and therefore may have been overlooked by the observer. It is quite possible that, long before our present patient complained of defective vision, there may have been considerable diminution in the range of his visual field, which might have been readily demonstrated by the use of the perimeter. Considering the constant enlargement of the pineal body, observed in these cases after death, and bearing in mind the very close relation of this structure to the optic tracts and chiasm, it will always be well to test the fields of vision in suspected cases. I think, further, that we shall find this test an early means of diagnosing the disease from any other.

Very little has been done in the way of treating this affection. It does not greatly tend to shorten life, and the patient may live, after the onset of his trouble, twenty or thirty years. The fact that the thyroid has in some instances been affected has led to the use of that body, or its extract, in the treatment, but with little, if any, benefit. Again, working on the supposition that the change in the pineal body may alter or diminish its activity, in perhaps the same way as the changes observed in the thyroid in cases of myxœdema, diminish or alter the activity of that gland, has given rise to the suggestion that cases of acromegaly should be treated by the exhibition of the pineal body, or some extract containing its active

principle. I may say that, considering the lesion observed in the pineal body, that it has been invariably, as far as I can ascertain, a hypertrophy rather than an atrophy, one would look upon acromegaly as more analogous to Graves' disease than to myxœdema, and would, therefore, expect but little, if any, benefit from pineal feeding.

We have already begun by feeding this patient on thyroid and an extract of the pineal body. Although the various measurements of head and hands, etc., and his general weight, have all diminished, yet the sight, especially of the right eye, is rapidly growing worse. Therefore, although I shall continue the treatment by these extracts to a certain degree, I shall also give him the additional chance of relief, and attempt to save what sight is still left to him, by treating him as an ordinary case of cerebral tumour, and give him iodide of potash with mercury.

NOTE.—The case, after being so treated for several weeks, was somewhat improved. The measurements of the head and hands were all diminished; the weight lost was nearly two stones. The fields of vision had slightly enlarged; but whatever improvement there was, only took place in the "nasal" fields, he never regained any "temporal" vision. His general health remained quite good.

A LECTURE

ON

TRACHEOTOMY AND ITS AFTER-TREATMENT.

Delivered to the King's College Medical Society,
January 17, 1896,

By H. LAMBERT LACK, M.D., F.R.C.S.,

Surgeon to the Throat and Ear Department, Children's
Hospital, Paddington Green;

Assistant-Physician Throat Hospital, Golden Square;
and Demonstrator of Surgery at King's College.

THE remark usually made in introducing this subject is that tracheotomy, however simple at times, may under unfavourable circumstances

present as great difficulties as any operation in surgery; and the mere record of the dangers and mistakes which have occurred in its performance are sufficient to convince us of this fact. However, the indications for the operation are oftentimes so imperative and urgent, that we must all be prepared to undertake it at a moment's notice, and under any circumstances, however adverse. I cannot possibly go fully into such a large subject, but as briefly as possible I will review the indications for opening the wind-pipe and the chief methods of doing it, with a short account of its dangers and the methods of avoiding them.

The indications for tracheotomy are mainly two. It may be required in certain operations about the mouth, nose, pharynx, or larynx to prevent blood or other fluids entering the trachea, to facilitate manipulation, etc.

The other and more usual indication for tracheotomy is to relieve immediate or prospective dyspnoea due to any form of stenosis of the upper part of the trachea, the larynx, or the parts above. Of the signs of this dyspnoea the most reliable one is recession, during inspiration, of parts of the chest walls, viz., of the lower part of the sternum, the lower ribs, the intercostal spaces and the supra-sternal and supra-clavicular fossæ. This sign is very definite of obstruction somewhere, and we must next determine its site.

If obstruction arises from morbid conditions in the mouth and nose or pharynx, or such causes as a large tumour in the neck producing pressure, this is at once apparent. If the larynx is the seat of obstruction, as is most common, we have two important symptoms quite apart from laryngoscopic aid, viz., the large up and down excursions of the larynx and the peculiar stridor. These movements of the larynx and trachea are a very marked feature of all severe cases of laryngeal stenosis, and may prove a serious nuisance in the performance of tracheotomy. Laryngeal stridor is also a quite characteristic symptom. It is a sound, once heard, must be remembered, and particularly never mistaken for snoring or stertorous breathing. If these two symptoms are present stenosis may be certainly diagnosed in the larynx or upper part of the trachea that is relievable by tracheotomy. These symptoms are said not to be present in tracheal stenosis, but I have carefully observed and noted them in two

cases of stricture of the trachea, and I can see no anatomical reason for their absence.

In cases of tumours, such as malignant disease in the mediastinum, aneurysms, etc., pressing on the trachea, these laryngeal excursions are absent, the trachea being fixed by the growth, and stridor is absent or quite different from laryngeal stridor. This is very important, as, of course, dyspnoea from such causes is not amenable to tracheotomy. These tumours, however, not unfrequently cause dyspnoea by involving both recurrent nerves, and so giving rise to bilateral abductor paralysis. This, of course, gives rise to signs of laryngeal obstruction, and requires tracheotomy.

Care must also be taken to exclude the dyspnoea of asthma, where both the above signs are absent, and, in addition, the difficulty is *expiratory*.

Cases of *broncho-pneumonia* may be more difficult to distinguish. With the usual signs of this disease, there is dyspnoea, cyanosis, a rattling in the throat, and often some recession of the chest walls, especially when complicated by collapse of the lung bases. There is, however, no stridor, and the recession of the chest is not nearly so marked as in cases of laryngeal obstruction with a similar amount of distress.

I shall now pass on to consider a few of the conditions which may give rise to obstruction of the upper air passages, chiefly with regard to the time at which tracheotomy should be performed. In cases of malignant disease, knowing full well that tracheotomy must come, we do it as soon as any dyspnoea appears, in order to operate in the most favourable circumstances. In cases of gumma of the larynx we first try if scarification and sucking ice will allow time for iodide to act: and similar treatment is adopted in oedematous affections. But in either case we operate if there is severe dyspnoea or if the above remedies do not afford immediate relief. In cicatricial stenosis, on the other hand, it is best to operate as early as possible. In cases of tubercular laryngitis we postpone the operation as long as possible or until dyspnoea threatens life, firstly, because tubercle has a great tendency to break down and ulcerate, and thus relieve the obstruction, but mainly because tracheotomy deprives the patient of the power of coughing efficiently, and thus

adds a very grave danger to his disease. In cases of wounds of the larynx or trachea or fractures of their cartilages it is better to perform tracheotomy at once. If obstruction be not immediately caused it will shortly ensue as a result of inflammatory swelling, and, meanwhile, blood may be entering the lungs. In severe cases of spasm of the glottis, it is well to have everything in readiness for tracheotomy, but operate only *in extremis*, as a fatal result but very rarely occurs. In cases of bilateral abductor paralysis an operation is not always necessary, and I would never advise it in infants or in very old or disabled people, unless really suffering from dyspnoea. A baby about six weeks old is attending here, which, in spite of marked recession of the chest walls and stridor lasting some weeks continuously, is fairly well-nourished and comfortable. It stands by far its best chance of recovery without tracheotomy. A year ago I showed to this Society a worn-out old man suffering from this affection, due, as is most common, to *tabes dorsalis*. He had as much breath as he required for the work he could do, and similar cases are not rare. I mention this especially because some teachers of anatomy are accustomed to point out that the posterior crico-arytenoids, the abductors of the cords, are the most important muscles in the body, their complete paralysis causing instant suffocation, and this opinion is also stated in many works on medicine. As a matter of clinical experience these cases not very uncommonly show no sign of distress.

In cases of diphtheria in children tracheotomy should be performed as soon as it is evident from the peculiar cry and cough that the larynx is invaded. Laryngeal obstruction, owing to the small size of the glottis in children, will almost certainly come on soon; but if we delay the operation till then we may have to perform it in a hurry, and the difficulties and dangers of an always sufficiently difficult operation in children will be increased, whilst the child is exhausted by its efforts. Also, by timely operation and special treatment it may be possible to prevent the spread of the membrane down the trachea. For the latter reason early tracheotomy is also to be preferred in adults, although here there are not the same dangers in delay. How far these views will have to be modified by intubation and antitoxin treatment I have

not experience to say. It is, I believe, a rule of our distinguished President to have his tracheotomy instruments at hand during every case of anæsthetisation. The necessity for this caution has been impressed on me by the following case:—After having removed some post-nasal adenoids in a young girl, the patient, after bleeding had ceased, and she was apparently coming round, was placed on her back preparatory to being removed to the wards. Suddenly she vomited and became cyanosed. She was quickly rolled on to her face, and her colour improved. But she soon became blue again, and artificial respiration was carried out. This for a time was successful; but again the patient became blue, and, to my alarm, in spite of great efforts, no air could be made to enter the chest. I pushed my fingers as far as possible into the throat, but could feel nothing; the face by this time was quite black, and the case desperate. The patient was hastily replaced on the table. I opened the trachea, and pushed my finger into the larynx. This caused a slight reflex, and a cannula was at once inserted. With slight artificial aid breathing was rapidly re-established, and the patient recovered. She owes her life entirely to the fact that tracheotomy instruments were at hand.

There are, of course, some rare conditions not mentioned in the list I have made, for which tracheotomy may be required. The following are illustrations:—A child of 4 was brought to Paddington Green, having been seized with great dyspnoea whilst eating her dinner. Tracheotomy was performed without success. At the post-mortem a large bolus of meat was found in the œsophagus, and the trachea was compressed between this and an enlarged thymus gland. Not long ago, at King's, a boy was admitted with a fractured jaw. A splint was applied, and his mouth closed for breathing purposes. He had adenoids, and could not breathe through his nose; therefore, if his mouth was to be kept closed tracheotomy was necessary.

I must now allude to a few conditions in which tracheotomy has been done where it was not required. It has probably been done most often for bronchitis and broncho-pneumonia in children, and I have already pointed out how this mistake may be avoided. Of course obstructive laryngitis may, in rare cases, co-exist with these conditions,

and really require tracheotomy, a fact which may prove of much assistance to anyone who should make the mistake, especially as œdema of the larynx is not well marked post-mortem. I have done tracheotomy at the request of two physicians for œdematous laryngitis occurring in the course of croupous pneumonia. I was once entirely deceived by a case of malingering. A young man was led into Golden Square leaning on his companions. He had great laryngeal stridor, and was apparently much distressed. He choked at all attempts at laryngoscopy. After two hours, during which he sat up in bed and continued this stridor, I did tracheotomy. He made a surprisingly rapid recovery, and returned home in about ten days with a normal larynx. I heard afterwards he was a deserter from the army, and had been a hospital orderly and thus had learnt his tricks. He acted his part well, submitted cheerfully to tracheotomy without an anæsthetic, and the idea of shamming never occurred to me at the time.

I have heard of cases where tracheotomy has been performed apparently for the stertorous breathing of cerebral compression. No less than two cases are known to me where tracheotomy has been performed in cases of cerebral abscess; in both instances the patients were deeply comatose, blue, with *stertorous* breathing. The real difficulty in these cases apparently was that the tongue was allowed to fall back, and tracheotomy was done without making any attempt to remedy this condition. In two cases of fracture of the base of the skull the operation was done for similar reasons, and in yet another case it was performed for a bullet wound of the brain.

There is another danger we run in indiscriminately operating. A man, apparently asphyxiated, was brought into a London hospital late at night by his half-drunken companions. The house-surgeon seized the nearest knife and skilfully performed tracheotomy, but could not resuscitate the patient. His friends departed greatly impressed by this proceeding which they had witnessed, and related that they had taken their dying friend to the hospital, where his throat had been cut. They were firmly convinced it was scientific murder.

I have quoted these cases fully because they warn us of the ridicule or even danger we encounter in rashly performing tracheotomy before we have even attempted to determine the exist-

ence of obstruction, or to relieve it by simpler means.

I have already alluded to the choice of time for operation. Tracheotomy should always be done as soon as its necessity is apparent, and it must be remembered that in the most chronic cases, when once slight dyspnœa has occurred, a violent paroxysm is liable at any moment to end the days of the sufferer.

We now pass on to the question of an anæsthetic. After the first incision the operation causes remarkably little pain, and in adults local anæsthesia, preferably freezing the skin, is all that is required. I have operated on many adults, including women, in this way, and they never complain of the pain. Again, for general anæsthesia the recumbent position is necessary, and we prefer the neck on the stretch. This position may seriously increase the dyspnœa, especially when the action of the accessory muscles of respiration is cut off; a spasm may occur, or the patient struggle violently. Under such conditions the operation may have to be undertaken at once, and at all hazards; and, among other dangers, blood may enter the trachea, the patient cannot aid us by a timely cough, and his life is in grave danger.

In children, and especially in acute diseases, on the other hand, chloroform appears reasonably safe. Even if not quite free from risk, there is probably less danger in giving it than in attempting a difficult operation on a struggling infant. Of course, where cyanosis is present, the child is already anæsthetised, and no chloroform is permissible. Giving chloroform to adults in these cases is, I believe, a wrong custom, and fatal results have occurred. I think all who have experience of the operation without anæsthesia will agree on the ease and confidence with which it is undertaken.

Having determined to open the windpipe, we have the choice of three classical operations:—laryngotomy, in which an opening is made through the crico-thyroid membrane, high tracheotomy and low tracheotomy, in which the trachea is opened respectively above or below the thyroid isthmus; and we must consider briefly the advantages and disadvantages of each method. For laryngotomy it is claimed that the parts being so superficial, it is the least dangerous and easiest of all. It has, however, many disadvantages. The

crico-thyroid space is small, and a specially shaped tube is required. The integrity of the larynx is impaired, the voice may be permanently injured, the tube is extremely uncomfortable to wear, and may produce ulceration or necrosis of the laryngeal cartilages. Again, the opening is too close to the seat of disease, and in such cases as malignant disease of the larynx, a second operation lower down will probably be required. From the small size of the larynx the operation is out of the question in children. I do not think we do right to perform a bad operation because it is easy, and therefore I would never do laryngotomy.

A low tracheotomy possesses great advantages. It is as far as possible from the seat of disease, it does not interfere with the integrity of the larynx, and the tube is worn with the most comfort. Its disadvantages are simply the difficulties of the operation, the longer time required for it, and its alleged greater danger. Similar advantages, in less degree, may be claimed for the high operation, which is also a more simple and rapid procedure. I would suggest, then, that low tracheotomy is indicated whenever it is likely that the cannula will be required for a long time, and especially in such cases as malignant disease, provided there appears to be time to complete the operation deliberately. In this case the dangers are no greater than those attending the high operation.

Where there is any urgency, and especially if only a temporary opening will be required, high tracheotomy should be chosen because of its greater simplicity and the rapidity with which it may be completed if need be.

The steps of the operation in the adult are briefly these. The patient should sit up in a suitable chair facing a strong light. The neck being shaved, etc., is extended as far as possible, and the chin placed in mid line with the sternum. Having frozen the skin, insert the knife at the upper border of the cricoid, and sink it deeply in until it meets the cartilage. Then cut vertically downwards for about $1\frac{1}{2}$ to 2 inches in the mid line. It is of great importance to make this first incision deep enough. Our only guide in the operation until the trachea is felt is the mid line of the neck, and while we adhere to this guide we are safe. If we make a small incision, insert retractors, sponge and look for divisions between muscles; we are very apt to get out of the mid

line and lose our way—an accident impossible with care whilst making the first incision.

We next feel for, and carefully define the cricoid cartilage, and make a transverse incision along its lower border, dividing the dense fascia which holds up the thyroid isthmus. By inserting the handle of the knife in this transverse cut we easily lift the isthmus downwards and expose the three upper rings of the trachea. These are now thoroughly bared, and all hæmorrhage arrested. The trachea may be now fixed with sharp hooks, one passed through on either side of the mid line, pulled well forward, and opened by cutting from below upwards. The orifice is held open by the hooks and the tube inserted.

A more simple procedure is simply to steady the trachea with the left index finger, and open it, then pass the finger into the trachea, and insert the tube as the finger is withdrawn.

In children, and especially in cases of diphtheria, a modification of this procedure may be recommended. When the thyroid isthmus has been exposed, define it, and clamp it on either side with a Spencer Wells forceps. These forceps arrest any hæmorrhage which might arise from division of the isthmus, serve also to steady the trachea, and to hold it open after incising it. On dividing the isthmus four or five rings of the trachea are bared, and also the trachea comes forward, easily lessening the depth of the wound—a great advantage in children. This method gives us plenty of room to inspect the interior of the trachea to remove any membrane which may be present, and afterwards to pack it above the tube with a view of arresting a downward spread of the disease. In all cases of diphtheria it is well to hold open the trachea, and encourage coughing before inserting the cannula. It is advisable also to be on your guard when opening the trachea, because of the violent cough excited, and the contents may come in your face.

In performing low tracheotomy we make a deep incision as before, but a little lower in the neck. This incision should be deep enough to find the interval between the muscles. After this one cut we use chiefly the handle of the knife, and as gently as possible work our way down to the trachea, constantly ascertaining, by means of the finger, that we are proceeding in the right direction. It is best to cut as little as possible, and then

always to cut upwards, thus avoiding any danger of wounding a large vessel at the root of the neck.

The trachea, being well bared, is steadied and opened as before. In consequence of the looseness of the tissues in front of the trachea in this region, the operation is usually a surprisingly easy one to perform. However, it must be remembered that the veins met with are large and thin walled, and may easily be torn by the handle of the knife if too much force is used; therefore, it is always well to have a free incision to work in.

In cases of extreme urgency, where the patient is struggling for breath, with all his accessory muscles acting vigorously, and in spite of this can get in so little air that he is cyanosed, other methods must be employed. In such cases the first touch of the knife causes the patient to fall back as if dead. This occurred once to myself. Now, to open the trachea immediately, as must be done, with the turgid veins all bleeding, is a matter of great difficulty and danger. In these cases and, of course, in all patients apparently dead of asphyxia, it is, I believe, safer to operate by the following method.

Seize the larynx firmly with the left hand, forcing the thumb and index finger, which point downwards, as far as possible behind it. By this means the windpipe is steadied and pulled forward, and the skin rendered tense. Now enter the blade of the knife straight down into the trachea about the level of the first and second ring. This must be done carefully, and as soon as the point is felt to be in the cavity of the windpipe cut carefully upwards for about half an inch. Withdrawing the knife, insert the finger into the trachea, and as the finger is withdrawn insert the cannula.

The steps of this operation are so simple, and it can be done so quickly that we have no need for hurry with it in even the worst cases, and therein lies its safety. It is obviously much better than any hap-hazard method of sticking a knife into the first available part of the windpipe, or missing it altogether, and wounding other structures. I have myself performed it three times with success, and I think it should unhesitatingly be employed in cases of great urgency in preference to any of the longer methods. Laryngotomy in one incision offers no advantages over this, and is, I am sure, no less difficult and no less risky to perform.

The one point in its performance to be borne in mind is to insert the finger into the trachea as the knife is withdrawn, otherwise great difficulty and delay may occur in inserting the tube. St. Germain (*Gaz. des Hôpitaux*, January 15th, 1881) reports that he has done 227 tracheotomies in a similar manner without any grave accident. He recommended that the knife should be entered above the cricoid, and the incision made vertically downwards.

This method of operation requires no special instruments. A knife of some sort is always obtainable, and, in the absence of a tube, the wound may be kept open by the handle of the knife, or by a retractor improvised, if need be, from a hair-pin, a piece of stiff wire, or anything that comes to hand. Various trocars and trocar-cannulas have been invented for use in these urgent cases. The obvious objection to all of them is that they would not be at hand when wanted, and even if I had them I would prefer to use a knife and a tracheotomy tube.

We will now review some of the dangers or mistakes which may arise in these operations, and the methods by which they may be avoided.

In young infants, where the larynx is ill-developed and difficult to feel, the mistake is often made of operating too high up. In this way the larynx has been opened, or even the thyro-hyoid membrane. This mistake may be avoided by care, the cricoid cartilage is nearly always distinguishable, or, in any case, it may be prevented by the excellent rule of making the incision extend downwards to within one finger's breadth of the top of the sternum.

The difficulties arising from a too small wound in the skin or in the trachea are as obvious as is the remedy for them.

Again, the mid line of the neck may be missed, or the trachea, when very small, as in infants, hooked aside, and the operator cut down on to the carotid arteries, the œsophagus or even the spine. This mistake has frequently occurred—I know of two men of world-wide reputation who have made it—and yet it is one which can easily be avoided by a little attention and care. In the first place, allow me again to emphasize the necessity of making the first incision carefully in the mid line, and deeply. Then we have no bother in hunting for the division between

muscles and missing it, and when retractors have been inserted we can feel the trachea, which ever after serves as our safe guide.

In this operation arterial hæmorrhage gives no trouble unless we wander from the mid line and open the common carotid. If we should meet with a *thyroidea ima* in performing the low operation there is no difficulty in dealing with it with a sufficiently free incision. The chief bleeding is venous, from the numerous large thin-walled veins often much distended with the dyspnœa, which we encounter mostly in the lower operation. These may be avoided, in part, by cutting as little as possible, and using the handle of the knife as gently as possible. The large ones must be dealt with, but bleeding from small veins may be neglected, as it will cease as soon as the trachea is opened, and free respiration established. Especially is this the case when no anæsthetic is employed and the patient can aid us by coughing up any little blood which may enter his trachea. Two other points must also be attended to. In using the knife in the low operation, always cut from below upwards so as not to endanger a highly innominate; and, secondly, take great care to see that the tracheal rings are well exposed so as not to open a vessel when incising the wind-pipe. No difficulty will occur in opening the trachea if it is transfixd with hooks, as before mentioned, and pulled well forward. The danger of wounding its posterior wall may be avoided by then shortening the blade of the knife, by pressing the index finger firmly against the blade within one half-inch of its point, and steadying the hand by resting the wrist on the sternum. Otherwise, not only has the posterior wall of the trachea been injured, but even the œsophagus and spine.

Another difficulty somewhat rarely met with arises from the ossification of the tracheal cartilages. In such cases a bone forceps or a small finger saw will be required—and the latter may, perhaps, be added to the list of instruments required for tracheotomy in patients over 50 years of age.

I have pointed out the methods by which the cannula may be safely inserted, either by transfixing the trachea by hooks before opening it, or by inserting the finger after it has been opened. If hooks are not used and the finger is not inserted, the cannula is often pushed down into the

neck in front of the trachea, especially if too small a tracheal incision has been made. This forms a track in which inflammatory products may accumulate, and subsequent cellulitis may be produced. But the great danger at the time is that the dyspnœa continues, and blood is being sucked into the tracheal wound. This blood can easily occlude the already obstructed larynx, and unless the tube be rapidly inserted the patient is likely to die on the table, an accident I have known occur.

The test that the tube is well in is, of course, the free passage of air through it. If the tube is really in and air does not pass through, then the obstruction must be seated lower down, and you must try what may be done by longer specially made tubes such as Kœnig's.

In cases of diphtheria, the tube, although in the trachea, may be blocked by membrane, or, it is said, pass in between the membrane and the wall of the trachea. This should never occur as, in such cases, the trachea should be freely laid open and all membrane cleared out before one thinks of inserting a tube.

Having completed the operation, the wound is dressed with a little iodoform, and a piece of gauze arranged around the tube. The patient is then returned to his bed in the ordinary ward, and no tents or steam kettles, etc., are used. The cases do just as well and, I believe, better without them. On the second or third day the patient is allowed up in the ward, and usually returns home, wearing a permanent tube, within a week. Of course, this only applies to simple cases of tracheotomy; where the patient has diphtheria or any other serious disease, it must be modified. Such cases come, however, under the care of the physicians, who will, of course, keep them in bed, put them in tents, steam them, and give them the latest remedies.

In diphtheria, the tube should be removed every 24 hours, and thoroughly cleansed by boiling it in a strong solution of soda or Hudson's soap. Before inserting another it is well to hold the trachea open with retractors for a few minutes, and encourage the patient to cough. In the intervals between this, removing the inner tube and cleaning it in a similar way is all that is required. If the patient has much trouble in coughing up tough mucous pellets a warm solution of soda

(gr. xx to the ounce) may be dropped down the cannula, two or three drops at a time. No feathers or contrivances of that kind can possibly do good, although quite capable of doing harm. In those terrible cases in which the membrane has spread down the trachea below the tracheotomy, these soda drops may also be tried. In one such case, in a child of 3 years, three violent attacks of suffocation occurred in two days, and each seemed likely to be fatal. In each attack I injected this soda solution repeatedly in half-drachm quantities, down the trachea, each attack subsided in about half an hour, and the child finally recovered. I cannot say how far the result depended on the treatment, but recovery when the disease, in spite of tracheotomy, has spread down so far, is extremely rare. The soda acts by softening the hard or viscid mucus, and thus aids the expulsion of the membrane.

There is sometimes a little difficulty in swallowing, and especially in drinking after tracheotomy, and the surgeon is sometimes unnecessarily alarmed by seeing fluids coughed up by the cannula. It does not necessarily follow that he has opened the œsophagus, or that the tube has ulcerated into it. On the contrary, it is a not uncommon thing, and arises, I think, from the disease or swelling about the top of the larynx, mechanically preventing its complete closure; or from pain and stiffness about the wound in the neck, which prevents the larynx and trachea moving up and down so freely as is usual in swallowing.

In such cases, of course, feeding by tube must be adopted, but will usually not be long required. Test the patient's power of swallowing by rolling him on his side or sitting him up and giving him teaspoonfuls of water to drink. Should a little water enter the trachea it is not very harmful, and if he can swallow it easily, other food may safely be given.

Finally, we remove the tube as soon as it is no longer required—in children, especially, the sooner the better. In cases of diphtheria the tube may be removed a day after the membrane has disappeared from the throat, provided the voice is clear.

I must next allude to some of the chief dangers or difficulties which we meet with during the after-treatment.

In old people, especially where operation has

been too long delayed, syncope may occur, and is probably due to carbonic acid poisoning. For a similar reason diphtheria cases do not always rally from a late operation.

Broncho-pneumonia, and similar troubles, more usually occur in diphtheria and such diseases, and in children, and are not due to tracheotomy.

Secondary hæmorrhage is also rare, although I have had a case. I think the best treatment, if practicable, is to lower the patient's head to prevent blood running down his trachea, to put in a Hahn's cannula, and pack the wound with a styptic gauze. Failing this, keep the patient's head low, open up the wound as far as necessary, and seize the vessel. Secondary hæmorrhage is only dangerous from the blood entering the trachea; no big arteries having been cut, no large amount of blood is likely to be lost.

Emphysema is a rare accident, and is probably most likely to arise if the skin incision be very small or the trachea opening out of the mid line. Cellulitis is a dangerous, but, fortunately, a rare complication. It must be guarded against by not injuring the tissues by the rough use of the knife-handle or by injudicious attempts to insert the cannula. The wound also is usually best left open, and not stitched up round the tube. If cellulitis occurs, prompt treatment must be adopted to prevent its reaching the mediastinum. If the tube fits badly or is not properly tied in, it may be coughed out, and lie in front of the trachea. This accident is at once recognised by placing the hand in front of the tube. But I was once hastily called to an unfortunate woman in whom this accident had occurred and was not suspected. In her efforts to breathe she was actually drawing air down into her stomach, and naturally suffered from flatulence, which it was attempted to relieve by peppermint, and similar remedies.

Ulceration of the trachea will occur only from an ill-fitting tube. If a Durham be used, its depth must be carefully regulated so that its extremity is free in the lumen of the trachea. The shape of a Parker is also specially designed to ensure this. An ordinary curved tube, such as the bivalve, must necessarily impinge on the anterior wall of the trachea, and cause ulceration, as in this specimen I now show you. It is the trachea of a child 2 years old, dead of bronchitis six months after tracheotomy for diphtheria. The

tube had to be retained because of a stricture of the larynx, as you see here, due, I believe, to the operation having been performed too high up, the cricoid cartilage being divided.

A properly fitting silver tube, such as Durham's, may be worn for years without discomfort. I have seen many children wearing them three or four years without any trouble, and ultimately able to do without them. Adults will wear them for years. I have known cases where the tube has been retained 10, 15, and even 20 years, and apparently with no ill-effects.

There are, however, some inconveniences in wearing a tube. The chief of these is that the cough is rendered inefficient for want of a glottis to be closed. For this reason bronchitis becomes dangerous, and in cases of phthisis the secretions accumulate in the lungs, and choke the patient, or general tuberculosis ensues. In three cases of phthisis, in which urgent dyspnoea compelled tracheotomy, all died within five weeks from the increased rapidity of the disease. Therefore in such cases we postpone the operation as long as possible.

The open mouth of the tube, of course, allows cold air and dust, etc., to enter the trachea directly, but the mucous membrane apparently accommodates itself rapidly to this. A few layers of gauze worn in front of the tube might, however, be well, to prevent any gross particles entering. Lastly, tracheotomised patients, for want of a glottis, cannot fix the chest, and are consequently incapable of much muscular exertion. This, however, is apparently partly overcome in course of time.

We sometimes meet with difficulties when we wish to remove a tracheotomy tube. The air striking on the cords apparently causes a certain amount of spasm, and the child is frightened and struggles until the tube is replaced. A little gentle persuasion may overcome this; if not, the inner tube should be removed and corked, and the unsuspecting infant thus deceived. If there is then dyspnoea, it may be that true abductor paralysis is present. This occasionally follows diphtheria, especially, it is said, if laryngotomy have been performed. It must similarly be treated by corking the cannula for increasing periods of time. We may have granulations springing up round the wound in the trachea. These are

usually indicated by a little blood-stained sputum, and prevent the permanent removal of the tube. In one case—an infant of 15 months—after various unsuccessful attempts to remove the tube, I enlarged the wound, scraped out the trachea, and in a few days was able to dispense with the cannula. Or we may have to deal with a stricture of the larynx or trachea, a condition difficult enough to diagnose, and worse to treat. Excision and packing would probably offer the best chance.

Finally, I must say a few words about the choice of a tube, as on this much of our success must depend. A metal tube offers overwhelming advantages. It is strong, easy to clean and disinfect, it will stand boiling, and it has a relatively larger calibre. It should have a complete inner tube which must project slightly beyond the outer one, and be easily removable for cleaning. The collar should be movable, so as to fit comfortably during movements of the neck, and it should also be adjustable to the end of the wound. It should have a curve so as to adapt itself to the trachea, and not press on either wall. It must have no detachable parts liable to fall into the trachea, and finally it should be provided with a good pilot to facilitate its introduction. A Durham or a Parker fulfil these indications better than any other tube with which I am acquainted. For a permanent tube a Durham seems the most comfortable, while for diphtheria I have generally preferred a Parker.

A CASE OF NEPHRORRHAPHY

PERFORMED BY MEANS OF

PROF. VULLIET'S OPERATION.

By ALBERT CARLESS, M.S. Lond.,

Senior Assistant-Surgeon to King's College Hospital,
London.

JANE D., æt. 41 years, was sent to me by Dr. Lindow, of Plumstead, in October last, complaining of a painful swelling in the abdomen, and of severe and almost uncontrollable sickness. She was a married woman, and had had several

children, but none recently. Her previous history is unimportant, and, indeed, she had enjoyed very good health up to within four years. At that period she noticed a swelling in the region of the right kidney, which was sometimes evident, but at times disappeared entirely. It was not especially painful, although occasionally vomiting was induced by it. About six weeks before I saw her, the pain in the side became very much more acute, and the vomiting so distressing that it almost always followed the introduction of food into the stomach, and was frequently preceded by neuralgia of the right side of the head and face. A certain amount of irregularity was also experienced in the act of micturition, which was sometimes interrupted for hours, and then again it would become very frequent, the desire occurring every few minutes, and being accompanied by pain if not at once relieved. On palpation of the abdomen, a swelling, the shape and size of the kidney, was readily to be detected on the right side, moving up and down on manipulation, so that although it generally occupied the right iliac fossa in the erect position of the patient, it could be easily slipped back to its original normal situation in the loin when the patient was recumbent. Its mobility and contour could be more exactly ascertained when the woman lay on her face, and a distinct loss of fulness and resistance in the loin could then be detected. She was admitted to hospital on October 16th with a view to operation, and, as I had been anxious to make use of Prof. Vulliet's method, which I had seen described in the *Clinical Journal* of June 19th, I determined to make use of it on this occasion.

The operation was performed on October 21st, under chloroform. The patient was placed on the left side with a sandbag beneath the loin, and the usual incision made for exposing the kidney, viz., one extending from the outer border of the Erector spinæ from a point half an inch below the last rib, downwards and outwards towards the anterior superior iliac spine for a distance of four or five inches. The muscles and aponeuroses were carefully divided in this line so as to expose the retroperitoneal fat, and on tearing this apart it was soon clear that the kidney lay below the level of the wound in the iliac fossa. From this situation it was readily displaced and drawn upwards to occupy a position

along the outer border of the spine at the level of the wound. A silk sling was passed through it temporarily so as to keep it in this situation whilst the next stage of the operation was undertaken. This consisted in making a vertical incision about two centimetres from the spine, three inches in length, the centre being opposite the first lumbar spinous process. After dividing the subcutaneous fat, a thin aponeurosis, viz., that of the Latissimus dorsi, came into view, and this was also incised vertically. One had now exposed to view on the outer side of the wound the muscular belly of the Erector spinæ, and on the inner side, close to the vertebræ, two or three long rounded tendons belonging to the Spinalis dorsi. The tendinous slip which was inserted into the first lumbar spine was selected, and carefully isolated from its surroundings by inserting an aneurysm needle beneath it, and working it up and down sufficiently to enable the index finger to be passed under it. Traction was now made upon it so as to drag down its upper portion, and without much difficulty the upper attachments of the tendon were torn through, and about ten inches of it brought into view, the upper end having still a certain amount of muscular tissue attached to it, the lower end still remaining connected with the lumbar spinous process. After trimming up the free end and removing the redundant muscular substance, a passage was burrowed through into the former or renal wound around the outer margin of the Erector spinæ, and through this track the free end of the tendon was passed. Two small transverse incisions were now made through the capsule of the kidney, one at the upper border, the other near the lower, and a communication established between the two beneath the capsule. Along this passage the tendon was drawn, and its free end finally passed through the muscles on the lower side of the renal incision, and there fixed by being threaded once or twice through them, and also by being stitched to them by a few catgut sutures. There was plenty of tendinous tissue to effect this without any difficulty, and, indeed, one removed finally an inch or two off the free end, which was unnecessarily long. The kidney was now satisfactorily fixed and scarcely moved at all, even on respiration. To relieve the drag upon the tendon, one or two additional silk worm stitches were

passed through the renal cortex and the adjacent muscular tissues at both ends of the organ. Both wounds were now entirely closed by means of rows of buried catgut sutures, the skin being secured by a continuous silk stitch. No drainage was employed. Our usual antiseptic dressing of cyanide gauze covered by salicylic wool was applied.

The after-history of the case was not altogether free from disturbance, although the wound itself did perfectly well, and healed throughout by first intention. On the day following the operation the temperature was raised to 100.4° ; the patient was very sick all day, and complained of great abdominal pain and distension; on examination we found the belly tense and tympanitic, and with marked surgical emphysema, which extended over the anterior wall and into the loins. All feeding by the stomach was interdicted, and nutrient enemata ordered. On the 23rd the condition was much the same, although the temperature was lower; the abdomen was still very distended, and an enema of turpentine and soap and water was administered. This brought away a good deal of flatus, and relieved the patient considerably. Only two ounces of urine were passed normally, but on inserting a catheter twenty-one ounces more were removed. This condition of the bladder remained for some days, the catheter being required, whilst on the sixth day the urine was smoky, and later on bright red in colour, and exceedingly offensive. The patient was treated by means of alkalies and tinct. hyoscyami, and later on with boracic acid, and the urine speedily became normal. The wound was entirely healed and the stitches removed on the eighth day, although there was still present a good deal of surgical emphysema. The patient was allowed to get up on the sixteenth day, and went out of hospital at the expiration of three weeks from the date of operation. She complained of a certain amount of aching pain in the side, but not more than one would expect considering the nature of the operation, and the drag upon the tendon and newly-formed fibro-cicatricial connections which supported the kidney in its new position. Directions were given to her not to engage in any heavy lifting for some time, and to remain at rest as much as possible for some weeks.

It will be at once evident to those who read the

original description of the operation in this Journal that the tendon I made use of for the purpose of fixing the kidney was not the same as that which Prof. Vulliet is stated to have employed. We are told that he used a portion of the latissimus dorsi; this must be an error, since that muscle arises from the lumbar spines by means of a flat aponeurosis, and not by rounded cords. As stated above, it is one of the tendons of the Spinalis dorsi which must be looked out for and utilised. As soon as the lumbar aponeurosis has been divided, this structure comes into view, and the surgeon can have no difficulty in isolating the appropriate portion that he requires. Another difference in the proceeding that I adopted consists in the fact that I took no precautions against sloughing of the tendon, because I felt certain that no such result was in the least likely to follow. A piece of aseptic living tendon will quickly contract adhesions to the tissues which surround it if the wound is kept free from infection, and no further thought need be taken of it.

The occurrence of surgical emphysema was at first perplexing, as one did not clearly see where the retained air came from; but a little consideration soon threw light upon this occurrence. The kidney at the time of operation was lying in the iliac fossa behind the peritoneum, and as soon as it had been restored to its original position, the cavity from which it had been displaced became filled with air, and this one was unable to expel owing to the position of the patient on her side. When, therefore, the abdominal parietes were closed by sutures, a considerable quantity of air was imprisoned behind the peritoneum. This became expanded by the heat of the body, and gave rise in measure to the abdominal distension which was so troublesome during the first few days of the after-treatment. The compression which it underwent, owing to the vomiting which resulted partly from the effect of the anæsthetic, partly from the interference with, and stitches which were inserted in, the kidney, forced it through the parietes into the subcutaneous tissues, where it became very palpable. On another occasion I shall make every effort to get rid of the greater portion of the air before closing the wound in the parietes. Probably the insertion of a drainage tube for 24 hours would obviate the difficulty.

As to the operation itself, I cannot but think

very highly of it as a safe and secure method of fixing the kidney. On several previous occasions I have operated for movable kidney, and have been forced to the conclusion, which is shared by most surgeons, that simply suturing it to the posterior abdominal parietes is a proceeding which, whilst occasionally giving good results, is not to be considered as a trustworthy proceeding in every case. In some patients, especially amongst the young, it will answer admirably, as occurred in a young man on whom I recently operated, suffering from marked mobility, the result of an injury sustained whilst opening a gate on horseback. But where the patient is older and the reparative powers not so active, something more than simple suturing seems to be required. The displacement by force of a portion of one of the tendons from the back does no harm to the individual, and no inconvenience or pain is afterwards experienced from its avulsion; its presence beneath the capsule of the kidney seems to be similarly harmless. Certainly I shall not have the slightest hesitation in performing this operation on any further cases of movable kidney that present themselves to me, and I anticipate that the final results gained thereby will be much superior to those obtained from any other proceeding.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, LL.D., F.R.S.,

(Reported by J. T. CONNER, M.D.)

Multiple Exostoses.

A MAN, æt. 34, was the subject of this disease, in a severe form. The growths were of variable size, situated on the long bones, mostly at the epiphysal junctions. Those on the right side were the larger, and had evidently caused arrest of growth. That at the lower extremity of the right tibia was the largest of all, and both the leg and foot were much shorter than on the other side. The right ulna was also shortened, and showed a large growth at its lower extremity. It did not articulate with the carpus. The radius was much curved;

but the movements of pronation and supination were not interfered with. There were exostoses also on the lineæ asperæ and the bicipital grooves.

The patient stated that the growths were congenital, and continued to grow as he grew, and ceased when he did.

General Dermatitis following Ringworm.

The subject was a girl, æt. 9, sent by Dr. P. S. Abraham. She had been under treatment for some months at Blackfriars Hospital for *Tinea tonsurans*. A week ago she became feverish and ill; an erythematous rash appeared over the body, but especially on the abdomen and back. Desquamation of the palms and soles followed. These were now cracked, inflamed and desquamating. The skin in general was in a xeroderma-like condition, with furfuraceous scalliness. There was slight hypertrophy of the hair follicles, especially on the elbows and knees. Dr. Abraham could vouch for the fact that the present condition was quite recent. It was similar to the case he had showed at the Museum last winter, excepting that in the former there was no history of antecedent ringworm. The case might have been taken for a congenital condition, or allied to Devergré's lichen.

A Growth in the pre-cancerous Stage.

An old man, æt. 85, sent by Mr. Waren Tay, showed a small hard disc, with a depressed centre, which appeared to be about to break down, on the back of the right hand. It was slightly elevated, and of about the diameter of a pea.

The duration was two years.

Remarks by Mr. Hutchinson. This growth probably began round a hair follicle. It resembles in appearance the crateriform ulcer of the face, of which it is no doubt the equivalent, though it has not yet shown definite signs of malignancy.

A Peculiar Form of Circinate and Corymbiform Lichen.

A man æt. 25, sent by Mr. Hitchens, was the subject of a peculiar form of erythema. The eruption consisted of confluent lichenoid papules, which evidently commenced in the hair follicles. It was thickly distributed on the limbs and back, leaving the face, hands, front of the trunk and

genitals, quite free. On the back the papules were arranged in rings. There was no itching. The rash had appeared suddenly a month ago. He had not taken any drugs, and nothing in his diet which might be a cause could be ascertained. The tongue was clean, and there had been no digestive disturbance.

Mr. Hutchinson obtained admission for this patient into the London Hospital, where he remained some weeks under the treatment of Dr. Stephen Mackenzie. No definite diagnosis was given. The man attended for demonstration on a second occasion a month later; the eruption had much faded, but had not disappeared. Mr. Hutchinson stated that he could neither give the eruption a name, nor assign any plausible explanation of its cause. A portrait has been preserved.

An Unusual Form of Rodent Ulcer.

A man, æt. 79, sent by Dr. Sturrock, from St. Pancras Infirmary, showed on the front of the right forearm an area about equal to the palm of the hand surrounded by a bossy, elevated sinuous border. The enclosed skin was in a condition of very superficial scar, with the hair follicles preserved. Extending inwards, all round, from the edge was a flap of skin, which left the central part uncovered but crossed by a narrow bridge. A probe could be passed beneath the flap from half to one inch, and from here a slight discharge of pus takes place at times. The disease began as a small red papule, eleven months ago, which gradually increased for six months, and then discharged pus. It continued to spread at the edge, whilst healing in the centre. Also at one part the elevated border was slowly retrogressing, leaving a gap.

Remarks. Mr. Hutchinson had never seen a case which showed the peculiar appearance here present. The skin had split into layers like paper. The disease, evidently infective, was either lupus or cancer.

An Urticarious Eruption of Peculiar Type.

A boy, æt. 7, sent by Dr. Walker, showed a general erythematous eruption, in the form of coalescing rings, about the size of florins. The edges were very slightly elevated, and of a deeper

red than the enclosed skin, which was becoming stained. The rash first appeared ten days ago on the face and neck as "heat spots." These, by spreading at their edges, and fading in the centre, produced the circinate condition now present. The palms were affected early. Some small vesicles had appeared on the ears, cheeks and chest. The itching was so intense that he scratched himself until the skin bled. The general health remained good. He had not been eating fish. There was a history of a former attack some years ago.

Remarks by Mr. Hutchinson. Urticaria differs from other pruriginous eruptions in that the patient may scratch it as much as he pleases without doing any harm. A question was raised as to whether the case should be diagnosed as erythema multiforme rather than urticaria, but the latter was preferred.

Chancre of the Lip.

A girl, æt. 21, sent by Dr. MacGeagh, had an indurated sore of the lower lip on the left side, with bulging of the prolabium. There was a deep crack in the middle. The left submaxillary glands were inflamed and adherent to the jaw, the right simply enlarged. The throat was normal, and there was no rash. She was decidedly anæmic. The crack in the lip had existed for five years, almost healing and relapsing. The present condition of inflammation dated from three weeks ago.

Remarks by Mr. Hutchinson. The fissure has no doubt afforded a ready mode of entrance to the syphilitic virus.

Symmetrical enlargement of Lymphatic Glands in the Neck, in middle life.

A MAN, æt. 43, showed a hard tumour about the size of a hen's egg projecting from beneath the anterior borders of both sterno-mastoids into the anterior triangle. That on the right side was movable, that on the left adherent to the muscle, but not to deeper structures. In the left axilla was an enlarged gland about the size of half a walnut. The throat and nose were normal. The patient stated that the lump on the right side was of nine, and the left of two months' duration; also that he had had syphilis several times.

Remarks. Mr. Hutchinson inclined to the view

that the disease of the glands was strumous. But the possibility that it was syphilitic was entertained. In tertiary syphilis lesions exactly resembling strumous enlargement of glands may occur, but they are rare. The symmetry was against the diagnosis of lymphosarcoma, but did not exclude it.

Scrofuloderma.

A boy, æt. 14, sent by Mr. G. W. Sequeira, had an oval elevated ulcer, about an inch in the greatest diameter, below the right ear. It commenced six years ago as a red pimple, and discharged pus at times. Near it on the cheek was a patch of brownish-red, about the size of a three-penny piece, leading to scar. This was of the same duration as the ulcer. There was a family history of consumption on both sides.

Remarks by Mr. Hutchinson. This case, though resembling lupus, differs from it in the fact that the subcutaneous tissue is the structure chiefly involved.

Elephantiasis of one leg; Strumous Ulceration (Bazin's Disease) of the other.

A man, æt. 23, sent by Dr. Barratt, had his left leg much enlarged, and in a condition of solid œdema, with papillary growths on the toes. At the knee were several scars like the "shilling scars" of rupia. On the front of the right leg were a number of indolent ulcers; and the tibia was decidedly thickened. The history was that six years ago the left leg was hurt by the boot, and four years ago by a cocoanut; the resulting inflammation leaving the enlargement when it had passed away. The disease of the right leg was also attributed to a knock from a stone some years ago.

Remarks. Mr. Hutchinson believed that elephantiasis was always produced by a succession of erysipelas attacks, each leaving behind some permanent enlargement. He did not consider that there was any re-infection, but that the germs of the first attack remained latent, until circumstances, such as injury, favoured their development. He suspected that syphilis was a factor in this case, because of the enlargement of the right tibia, and its aching at night, which do not occur in simple cases of Bazin's disease.

White Patch on the Tongue, like those which accompany Lichen Planus.

A man, æt. 37, showed an oval patch with a white rough edge on the right side of the tongue, near the tip. It was of two months' duration, and gave no pain or inconvenience. He was in robust health. By applying an antiseptic he could cause it to temporarily disappear.

Acro-scleroderma following Raynaud's phenomena.

A woman, æt. 27, sent by Dr. Little, had the skin of the hands glossy, shrunken, pale, and wooden. The fingers were pointed and had lost their tips. The changes were quite symmetrical. The little fingers had the last joints fixed in flexion. The loss of tissue was greatest at the tips of the index fingers. The left had entirely lost the nail. The face was dotted with stigmata, the features contracted, the lips thin and not covering the teeth. The skin could not be readily pinched, and when she smiled there was evident loss of mobility. The ears were affected. The feet were cold and dusky, but otherwise normal. The pulse was not abnormally weak. The disease commenced nine years ago in winter, when the patient worked at a florist's, and had to expose her hands to cold, by putting them in water. The fingers became dead and white, and then blue. As a child she had chilblains badly. These attacks continuing, the finger tips became sore and little black patches separated.

Remarks by Mr. Hutchinson. Though Raynaud's phenomena, followed by symmetrical gangrene, in the form of acro-sphacelus, are undoubtedly present, the case must not be considered a typical form of Raynaud's disease. Mr. Hutchinson objected to the term, as not only Raynaud, but subsequent observers, confused under that head a number of diverse conditions. The vascular changes with severe acro-sphacelus might exist, and yet no scleriosis, as here present, be produced. On the other hand, extensive scleriosis and acro-sphacelus might occur without Raynaud's phenomena. And the two conditions might co-exist, as in the present case. All the cases of acro-scleroderma which he had seen had occurred in women under middle age; and it was remarkable that all the museum portraits showed an exactly similar deformity of the little finger as the patient.

THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 12, 1896.

A LECTURE
ON
ADULT ANTERIOR POLIOMYELITIS
LECTURE I.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, May 22, 1895,

BY

W. R. GOWERS, M.D., F.R.S.,

Physician to the Hospital, and Consulting Physician,
University College Hospital.

GENTLEMEN.—The case which I have to show you to-day is in a special way instructive. We can indeed meet with no cases that are not instructive, or at least we should meet with none. If we do, the effect is due to our incapacity to receive instruction, not to the power of cases to give it. There are some, however, in which the information lies upon the surface for us to pick up; there are others in which it is beneath the surface, and consequently has to be sought for. But it is important that we should endeavour to acquire the habit of striving to obtain definite new knowledge from every new case we see.

The case which I desire to make useful to you to-day resembles most others, in that it illustrates that which is common, and also that which is uncommon. In most cases there are symptoms which you are able to recognize readily, symptoms with which you are already so familiar as to be prone to pass them by. Yet these are seldom the same in character and association; in different cases they vary sufficiently to make the process of discerning the variations a useful educational effort, and a source of fresh knowledge. Moreover, the familiar symptoms seldom fail to present, in their causation, problems differing more than do the symptoms. They often need careful thought, and then consideration constitutes fresh training in diagnosis. I cannot too strongly urge on you the habit of obtaining something new from

every case. You can do it if you will, and if you persevere until you have succeeded. Little labour is, however, necessary for this in the case before you.

The man's age is 47. There is nothing important in his family history; nor is there anything of special moment in his personal history. He probably had syphilis thirty years ago, but there is nothing in his present symptoms to indicate an association with that disease. It is seldom that any syphilitic process develops thirty years after the malady, whether it be a direct or indirect syphilitic process. That is an important practical consideration. There is a tendency to overrate the probable influence of syphilis. Because this cause has many consequences, whatever resembles those consequences in any way or degree, and follows the cause, is ascribed to it, even long after its occurrence. A period of thirty years after the primary disease brings most patients into the degenerative period of life. Some pure and primary degenerative processes resemble those that are the result of syphilis, but the syphilitic virus has then generally ceased to act and the causes of degenerative processes have become active and prominent.

One year ago, the patient suddenly noticed that his left hand was weak, so that he could not use his tools properly in his occupation as a carpenter. The left hand felt cramped and numb, and the same feeling of numbness has persisted. A few days later he noticed a tickling sensation at the bottom of the right foot. There are no persistent symptoms in the hand, and the disturbance that occurred a year ago has no obvious connection with his present trouble. Seven months ago he was suddenly seized with "severe pain in the back, passing round the left side to the front, and down into the left leg." This pain in the leg has continued in a less degree. It is now felt at the back, at the mid-dorsal spine, and passes obliquely round the abdomen to the front inguinal region. Formerly it is said to have passed down the back of the left leg. We cannot get more precise information about the early pains.

Pain in the back is very common. You are

familiar with lumbago, which, as you know, is pain connected with the muscles of the lumbar region; but lumbago, although common, is the most mysterious form of back pain that we meet with. We say, "It is a rheumatic affection of the lumbar muscles." In the same way we say that a patient with gout who has thrombosis in the femoral vein, with swelling of the whole leg, has "a gouty affection of the leg." This affords as much information as does the description of lumbago as "a rheumatic affection of the lumbar muscles." We have no distinct idea of what rheumatism of the muscles is; and lumbago, as rheumatism of the lumbar muscles, presents to us many strange features. It may be instantaneous in onset, and anything but instantaneous in disappearance. As long as it lasts, it is distinctly related to muscular action. The structures connected with the muscles seem to become so sensitive as to cause pain when mechanically influenced. Of the real nature of the affection nothing has yet been discerned. I think that no one has ever attempted to see, even through the microscope, what it is. And yet patients very often die from maladies such as pneumonia, the onset of which is marked by lumbago. "But," you may say, "could anything be discerned? It is simply rheumatism of the muscles and of their fibrous tissue." Possibly; at the same time there is this strange fact—that rheumatism of the fibrous tissue of the muscles may spread along adjacent fibrous tissues to the sciatic nerve, and in the sheath of the sciatic nerve may pass into an acute, most intense, inflammation, such an inflammation as could, unquestionably, be discerned as definite tissue change. Does not this suggest that there must be, in lumbago, a definite, actual morbid process not unallied with inflammation? Moreover, lumbago is not always lumbar; it may be felt over the sacrum, as was this patient's pain, and when felt over the sacrum it has exactly the same relation to movement as when it is in the lumbar region, and has far more tendency to pass down to the sheath of the sciatic nerve. It is a definite variety of lumbago, not differing, however, in its causation or course, except in its greater tendency to spread. I mention it because this patient's pain bore considerable resemblance to that form I have mentioned, which may be called sacro-lumbago, or sacralgia.

In him it was not a simple, transient thing. After the onset he lost the use of the right leg, from the hip downwards. Although the pain did not pass into that leg it passed into the left. Indeed, on close questioning, he is inclined to think that in this left leg it began in the calf, and passed up the leg, extending very rapidly (in the course of a few hours) up the back of the leg to the sacral region, and then developed there, being followed by the loss of power in the right leg. It increased during six days, so that at the end of that time he was unable to move the limb. But, except for the slight, subjective sense of "numbness" in the foot, there was no change in sensation in the limb, and we find none now in that leg or the other. But we find persistent loss of power in the right leg, and, as you see, there is much more than loss of power. There is conspicuous wasting. You can see, even from a distance, how much smaller the right leg is than the left. You perceive that he can flex the hip a little, but is unable to raise the foot. If I flex his hip for him and support the thigh, even then he is unable to extend the knee. Indeed, on the effort, I can discern scarcely any contraction in the muscles. The patient is also unable to flex the ankle; and can only slightly move the toes. If I flex the knee, and tell him to resist my effort to extend it, he is totally unable to do so. Flexion of the knee is one of the movements that are often not tested. It may be lost or lessened, and the change may escape detection. When I make the patient try to extend his ankle-joint against the resistance which I oppose by pressing on the sole, he is unable to exert any power.

This man's leg is manifestly devoid of almost all motor power, but sensation is unimpaired. Reflex action from the left sole is distinct, from the right sole is quite absent. The cremaster reflex on the left side is distinct, but is absent on the right. The abdominal reflex is not distinct on either side. The notes of the case say that there is a difference of five-eighths of an inch between the two calves, six inches below the lower border of the patella. But I would advise you not to measure in that way. There is no more fertile source of error than measuring the legs at a certain distance from a fixed point. In the case of the calves, there is no reason for adopting the method, because we always can find a place of maximum circumference. It may

not be at quite the same place in the two legs, but we can always get a maximum which we can compare with precision. $10\frac{3}{4}$ inches is the maximum girth of the right calf; and $12\frac{3}{4}$ that of the left. Thus, the difference is two inches instead of about half an inch, as it may be in health from the "dextral pre-eminence."

When there is wasting of the calf, the place at which the muscle is largest undergoes some alteration. If we merely measure each leg at a certain distance from the patella, we may fail to ascertain the actual difference. It is only thus we can explain the discrepancy between the measurement recorded in the notes taken only three weeks ago, and that which I have just made. I draw your attention to the point because it is a common source of error, and because the method that is accurate is much easier than that which is inaccurate.

Above the knee we meet with a much greater difficulty, because we cannot find a maximum in the gradually decreasing thigh. An instrument has indeed been made for the purpose, but such instrumental accuracy, excellent for precise scientific observation, is hardly within the range of general use. Most practitioners have to be content with what a well-known essayist once called "the practical service of imperfect means." We should never be content with imperfection when we can get perfection, but, if this is unattainable, we can often do much with means that fall short of it. If we cannot get maxima in the thighs, and cannot, with any precision, make measurements within certain distances of the knee, we can easily get a minimum. Minima reveal differences much less adequately than do maxima, but that which they do reveal is more significant. As a rule, just above the knee, the circumference of the thigh is a little less than it is over the knee itself, and much less than it is anywhere else up the leg. The muscular substance of the thigh extends down to the region of the minimum, and hence, there usually reveals a definite wasting. This is revealed only by a trifling difference, and that difference depends more on the change which has occurred in the recti than in the recti. But the small degree of difference is compensated by the precision with which it can be ascertained. The left thigh of this man is muscular, his vasti are well developed, and just above the patella, it measures $13\frac{1}{2}$ inches, while his right

thigh at the same place measures 13 inches. This difference is absolutely pathological.

When we find such marked wasting and weakness as this, the electric irritability of the muscles must be tested. Loss of muscular power depends upon disease somewhere between the cortical motor cells of the brain and the muscular fibres, and those of you who have been here often will be familiar with the conception that this path consists of two segments, disease of each of which causes quite different effects.

I indicate on the slate a pyramidal cell of the cortex, with its short branching processes—dendrons, as they are now called. They consist of fibrillæ, which join, and, passing through the cell, many of them unite to form the axis-cylinder process, or "axon," which extends far down the cord, and at last sub-divides. Its fibrillæ seem to end in contiguity with those of the shorter "branching processes" of a motor cell of the grey matter of the spinal cord, *i.e.*, the lower segment. To this pass also the fibrillæ from the posterior roots, and its "axon" is a motor fibre for a muscle.

In disease of the upper segment, the muscular nutrition is not greatly interfered with, and reflex action is increased; and that, in disease of the lower segment, there is muscular wasting, and reflex action is abolished or lessened. Therefore, whenever there is loss of power there must be disease of the upper or of the lower segment. When loss of power is accompanied by muscular wasting, and loss of faradic irritability, the disease is in the lower segment. In disease of the upper segment alone there is no extreme muscular wasting; nutrition and reflex action are intact.

Where there is loss of reflex action, to ascertain whether it depends on the sensory or motor parts of the arc, it is necessary to test sensation, and to test the nutrition of the muscles. Here, there is no loss of sensation, but there is wasting of the muscles; and, as you will presently see, we find the reaction of degeneration. Therefore, we have no reason to believe that there is any disease of the sensory part of the reflex arc, but, on the other hand, all the symptoms are accounted for by the disease of the motor portion. The importance of this you will see in a moment.

We will test the muscles first with faradism, and I will use for the purpose the best little battery of any kind with which I am acquainted. Those of

you who are not familiar with it may like to know that any practitioner can keep it in perfect order by himself for twenty years; that is saying a great deal. It is the small apparatus of Stöhrer, with a movable slip of zinc. There is a carbon cell, and the zinc for use is put in through an indiarubber diaphragm. Only the secondary coil should be used; it is drawn up over the primary coil by raising a rod attached to it, and the advantage of this is that the gradation can be as gentle as it is possible to make the movement of the fingers. Another advantage of this apparatus is that the hammer, by which the automatic interruption is effected, can be easily stopped or allowed to work while the rod can be raised or lowered by the thumb and forefinger of the same hand.

In testing muscles I prefer to hold the two electrodes in one hand, and apply them near together; this has the advantage, especially in the use of the constant current, that there is less diffusion than when they are far apart.

On the wasted leg you will notice the faradic current gives pain. If you want to avoid pain, and it is often important, move the hammer with the fingers, so as to use the isolated shock. This has, moreover, the advantage that it is more delicate. A greater length of coil has to be employed. But the chief effect on the sensory nerves seems to be due to the "summation" of the rapidly recurring shocks, which constitute what is called the faradic "current." You see instant contraction from the application of the faradic shock. Although the amount of coil used is greater than that employed in the "current," from which he shrank, it does not hurt him. In the anterior tibial muscles of the left leg the shock causes energetic contraction; in those of the right leg none. I leave the same amount of coil in use, and adjust the screw to let the automatic arrangement produce the series of quickly recurring shocks—the "current"—you notice how the patient winces. This may suggest to you how valuable the employment of the isolated must be in ascertaining the irritability of the muscles of children? In them it is most important to avoid giving pain. Once cause pain, and the battery is never even seen without harmful emotional disturbance. I need not test the other muscles; they would show the same result.

I told you that the voltaic irritability persists,

the faradic being lost; but the voltaic, instead of being excessive, as it usually is in such cases, is also lowered. When the motor nerve endings are degenerated, the muscular protoplasm usually reacts to the voltaic current more readily than in health, apparently in consequence of some slight nutritional change in the fibres. It is too slight to be revealed by any microscopical alteration in their aspect; but the fact that it develops gradually shows that it cannot be a mere effect of the loss of the nerve impulses; it must depend on nutritional change. But why should the voltaic irritability here be less than normal? In such cases of nerve degeneration we meet with diminished voltaic irritability of the muscle in two conditions. Months after the onset, if the paralysed muscles have not been stimulated by the application of voltaism, the irritability, at first increased, sinks below the normal, apparently in consequence of the entire absence of functional activity. That is probably the case here. But the voltaic irritability sometimes falls below the normal within a few weeks of the onset, and such early fall is then an exceedingly grave symptom. The diminution usually goes on to extinction of irritability, and the cause is clearly revealed by a microscopical examination of the muscles, which has been made in a few instructive cases of this class. Instead of the muscular fibres preserving, for the most part, their transverse striation, as in the cases in which their excitability is increased, they present extensive granular and fatty degeneration, so that in a large number all trace of striation has disappeared. These are cases in which the lesion is peculiarly irritative in character. The nutritional change of the motor nerves seems to share the special character of the spinal lesion, and the destructive alteration spreads from them to the muscular fibres, and leads to their disintegration. The nerve-endings are contiguous, but not continuous with the muscular protoplasm. The discontinuity does not prevent the nerve impulse exciting the muscular tissue to contraction; it seems to be a sufficient barrier to the proposition of simple degenerative changes, but irritative change seems to overleap it, and to spread from molecule to molecule in the muscle. In such cases there is little hope that any treatment will be effective. Stimulate the muscles with voltaism as you will, you cannot renew the excitability.

The irritability is here too low to allow me to show you the polar reactions, that is, to demonstrate at which pole, positive or negative, contraction occurs with a weaker current—that is, at which it first occurs if a very weak current is progressively strengthened. In many of the affected cases the excitability is greater to the positive pole than to the negative. It should not be so; the normal excitability is greater to the negative pole. But this alteration is by no means invariable; in disease of either the motor grey matter or the nerves the excitability may remain greater to the negative electrode. We do not know on what the difference depends, and we are not yet able to attach any significance to it.

The case presents other points of importance, but to this I must direct your attention at our next meeting.

A LECTURE ON MECHANICAL OR TRAUMATIC ARTHRITIS.

By W. ARBUTHNOT LANE, M.S.,

Lecturer on Anatomy, Guy's Hospital.

Now, Gentlemen, that we have plodded conscientiously through the anatomy and physiology of most of the typical attitudes of activity and of rest which we have been able to study as progressive actualities in great perfection and in the minutest detail in the skeletons of vigorous labourers and of such feeble subjects as have habitually assumed attitudes of rest the tendencies to change which exist during the assumption of any single posture of activity or of rest, I propose to consider separately the changes that take place in the joints of these skeletons, and to contrast with them other articular conditions which are produced mechanically, and which bear to them a certain similarity. The mode of production of the latter differs from the former in that it is due to force applied usually on only one occasion.

To you, who have carefully followed me through the varying changes which the several bones and joints undergo when the functions they are called

on to perform differ from the normal, I feel that I owe an apology for reconsidering, however briefly, material we have thoroughly threshed out, but I do so for two reasons. One is, that, owing to a want of familiarity with pressure changes and consequently with the normal physiology of the skeleton, you will constantly find surgeons describing the structural changes which you have seen to result from an altered physiology, and which are perfectly normal to the individual as being the products of disease. This I think due largely to the fact that the presence of any deviation from what is considered in anatomical works to be the normal form and type of bones and joints (in other words, the average or common type), such as partial or complete ankylosis of amphiarthrodial joints, the conversion of amphiarthrodial into arthrodial types, the eburnation and limitation of the range of movements of movable joints, the development of completely new joints, etc., invariably suggest to their minds the necessary presence at some antecedent period of some inflammatory condition, and they will talk glibly of their having been produced by tubercular or other form of inflammation, while occasionally, when such suppositions are too manifestly absurd they will fall back on the term "congenital," and speak of the appearance presented as an "abnormality." Gentlemen, I would warn you to avoid as far as possible this feeble mode of trying to avoid the careful consideration of a mechanical problem. The less keen the observer the more readily does he support himself with this prop. I need only mention this to you since you are already thoroughly familiar with the very definite changes in bones and in the intervening soft parts which of necessity follow upon variations in the manner in which pressure is transmitted through any part of the skeleton.

The chief blame for this want of knowledge on the part of the surgeon rests with the anatomist who ignores almost completely the physiology of the skeleton in his irregularly distributed treatises upon its structure. As I have shown you, it is exceedingly difficult to arrive at a knowledge of the functions of the skeleton by confining one's observation to the study of the so-called normal skeleton, and that it is only by the careful examination of the fixed and exaggerated attitudes that it can be done with comparative facility, perfect cer-

tainty and scientific exactitude. Each attitude admits of accurate examination and explanation.

The second reason is that changes in joints somewhat similar in appearance to those you recognise as being produced by the habitual assumption of attitudes of activity or of rest may result from the sudden transmission through them of considerable pressure as in the case of a forcible impact being sustained by the part. These changes, as you see from an examination of these specimens, possess only a most superficial resemblance to those we have previously examined, yet to the surgeon who is unaware of the manner in which the latter are brought about they are practically indistinguishable. As you are aware, the term rheumatoid arthritis is that which is almost universally applied to the majority of these conditions. Since it is one that your examiners will insist on your using, you must sacrifice your common sense and knowledge till you have passed beyond their control.

Of the pathology of rheumatoid arthritis we know at the present moment little more than enough to say that it resembles, in no manner, the conditions to which the term is applied almost universally. Dr. Wohlmann, whose name is familiar to us all at Guy's Hospital, has had several opportunities of examining cases of rheumatoid arthritis after death, and he has succeeded in finding what he believes to be the organism which is responsible for its production. I understand that his work on this disease will be in our hands very shortly. Several examples of this disease in infancy have been examined pathologically by my medical colleagues at the Hospital for Sick Children, but I cannot at this moment put my hand on the Transactions in which they are published.

In the Transactions of the Pathological Society, 1886, I communicated a paper entitled the "Causation and Pathology of the so-called disease Rheumatoid Arthritis and of Senile Changes," in which I pointed out that all the conditions which were described pathologically as being produced by the presence of this disease rheumatoid arthritis were produced mechanically, and I could not find in our museums any specimen of so-called rheumatoid arthritis which was not caused in this manner, and had obviously no casual relationship whatever with the disease rheu-

matoid arthritis as we know it clinically in the present day. My subsequent experience has served to strengthen the position which I took up at that time, and I have, in previous lectures, endeavoured, as far as possible, to familiarize you with the details of the facts and arguments upon which it was built. The term which I applied to these mechanical changes in the skeleton was one which suggested the mode of their causation, namely, "mechanical or traumatic arthritis," and you will, I think, agree with me that, being fully descriptive, it is a very serviceable and scientific one. At the same time I do not for one moment use the term arthritis as suggesting any inflammatory causation.

I am very pleased to find that my keenly observant and critical friend, Mr. Bland Sutton, has been able recently, by his observations on the skeletons of domestic animals, to satisfy himself of the truth of what I have explained fully to you, namely, that a variation or specialisation in the mode of pressure transmission through a joint, especially if this pressure is excessive, results in definite changes in the soft parts between the bones and then in the ends of the bones themselves.

I will now formulate a few statements regarding pressure changes in bones and joints other than those brought about by the exercise of considerable force abruptly to the part on a single occasion, and with each I will give you one or more references to conditions already fully described, and which serve as ample evidence or proofs of each.

1. That the so-called normal anatomy of a joint of whatever form remains normal only as long as it is called on to perform a usual combination of attitudes of activity and of rest.

2. That the habitual or frequent adoption of an attitude of activity or of rest produces a fixation of the attitude, which becomes exaggerated later. This results from changes in the joints, and in the growing subject it is modified largely by the consequences of unequal pressure exerted on the epiphysial lines in obedience to the general law which I formulated, viz.: that the rate of growth of bone in the several parts of an epiphysial line varies inversely as the amount of pressure transmitted.

The joint changes consist in the gradual com-

pression, absorption, and, finally, the complete removal of the soft parts in such places where an excessive pressure is transmitted, and in advanced cases the opposing bony surfaces may unite to one another by bone. On the margin of the bones which are compressed lips of dense osseous tissue are formed, and such adjoining growths readily unite.

The behaviour of any particular joint varies with its character and the peculiar function it carries out. While amphiarthrodial joints readily ankylose, in enarthrodial joints areas of articular cartilage are removed, the exposed bony surfaces are eburnated, and the form of the articulation is changed by the marginal deposit of new bone. For examples of these changes I would refer you to the joints of the porters, coalheavers, coal trimmers, shoemakers, sailors, charwomen, and other labourers, together with those of cases of lateral curvature, dorsal excurvation, knock-knee and flat-foot which we have examined.

3. That pressure exerted directly upon bones alters their form in a manner varying with the character of the pressure is shown by the sternum of the shoemaker, and the progressive section of the lamina of the fifth lumbar vertebra in the spondylolisthetic condition of the coal heaver, and by the divided fourth lumbar lamina in the fully developed coal trimmer, etc.

4. That excessive movement readily alters a joint from the amphiarthrodial to the arthrodial type. This is shown exceedingly well by the arthrodial joint which develops between the bodies of the lumbar vertebræ in the coal trimmer, the developing joint in the ossifying cartilage of the first rib, etc.

Perhaps, to those who have neither the time nor inclination to plod through all this detail the following description of the normal anatomy of a case of torticollis will explain a great deal. It was obtained from the body of an aged female which I dissected; I published the case in the Transactions of the Pathological Society, 1884. As the manner in which a varying physiology regarded as an altered mechanical condition has produced definite changes is obvious and simple, I will satisfy myself with a mere description.

Woman, æt. 60 years. Left sterno-mastoid represented by a narrow band of tendon. The face could be easily directed forwards, the chin

being a little raised by the movement. This movement apparently took place in the lower part of the neck, the upper part being comparatively fixed. It was a gliding movement, combined with some rotation around an antero-posterior axis. A large curve, with its convexity to the right, occupied the lower cervical and the dorsal spine. The normal convexity of the dorsal curve was increased considerably, and that of the cervical curve slightly.

The upper three cervical vertebræ were normal. The fibro-cartilage between the fourth and fifth cervical vertebræ was wedge-shaped, the apex of the wedge being to the left. Over it the left margins of the bodies of the vertebræ had fused by an osseous deposit. Union to a greater extent had taken place between the fifth and sixth vertebræ on the left side. The left articular processes and the halves of the laminae had fused together, forming one deep lamina.

The body of the seventh cervical was much diminished in depth, and there was much osteophytic growth at its margins. The anterior ligament which connected it originally to adjacent vertebræ was absent.

Lying on this and on the adjacent vertebræ, and behind the œsophagus, was a loose synovial cavity, which communicated with synovial spaces formed by the partial, and, in some cases, complete destruction of the fibro-cartilages above and below the body of the seventh cervical vertebra, so that, instead of the amphiarthrodial joints which exist normally in this situation, there were two of an arthrodial type, and allowing of very free movement indeed. The margins of the dorsal vertebræ presented much bossing, and many were more or less completely ankylosed by this dense bony material, viz., the sixth, seventh, and eighth, and the ninth, tenth and eleventh dorsal vertebræ. The deformity and inconvenience produced by the degenerated muscle was, to a very great extent, overcome by the formation of this very large curve, by the diminution in depth of some of the vertebræ and of the fibro-cartilages, by the alteration in the shape of the vertebræ, and by the formation of the remarkably complete arthrodial joints in connection with the body of the seventh cervical vertebra.

This case shows very well the effects of pressure alone in causing diminution in thickness of the

fibro-cartilages, and later, if there be much movement, the formation of articular cavities, while, with a greater limitation of movement, pressure causes, as well as absorption of the cartilage, the formation of osteophytic growths from the margins of the vertebræ at the seat of greatest pressure, and later, partial or complete fusion of the bodies of the vertebræ.

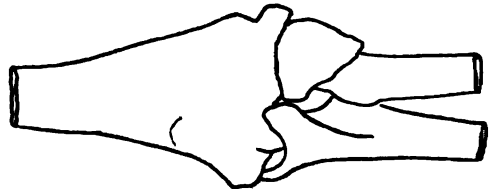
I propose now to show you some specimens illustrating the condition of mechanical or traumatic arthritis as brought about by the sudden and abrupt transmission of a considerable amount of force through a joint or joints on a single occasion.

I would just remind you of a fact to which I called your attention when we were considering the influence exerted upon dependent joints by such alteration in the mode in which pressure was transmitted through them by the unsatisfactory union of fragments when one or more long bones are fractured, a condition which almost inevitably results in a greater or less degree from the very imperfect and utterly unscientific manner in which these injuries are treated in the present day. It is that the changes which develop vary with the age and with the vigour of the subject. Up to a certain age bone, cartilage and synovial membrane are readily evolved, while later in life such compensatory changes in the form of bone do not result without destruction of articular cartilage, eburnation, and other frictional changes in the opposing bony surfaces, marginal deposit of bone, with increase in the bulk of the synovial membrane and of its secretion, etc., all changes coming clearly under the head of mechanical or traumatic arthritis.

I will now proceed to illustrate, by clinical history and by pathological specimens, several typical examples of this variety of mechanical and traumatic arthritis.

1. An elderly woman trips over a piece of orange peel, and falls heavily upon the extended palm. The lower end of the radius resists the breaking strain which it sustains, and the whole of the force which is transmitted along the shaft of the radius is impacted upon the radial head of the humerus, bruising the opposing surfaces of articular cartilage severely. After the accident the elbow joint becomes painful, the joint remaining more or less completely fixed in the position in which it sus-

tained the injury. After a time the tenderness in the part subsides, and the patient acquires some degree of movement, chiefly in the direction of rotation of the shaft of the radius, the range of flexion of the elbow joint continuing very limited. On examining the joint the head of the radius is found to be much altered in shape, and the radial head of the humerus presents much bony deposit upon its margins. The styloid process of the radius occupies an abnormal relationship to that of the styloid process of the ulna, being on a higher level than on the opposite side. The pathological condition shows a destruction of the articular cartilage covering the opposing surfaces of the radius and humerus with frictional changes and sclerosis of the exposed bones. A quantity of new bone is deposited around the head of the radius, and about the altered and limited facet on the humerus. A certain slight amount of change of a similar nature has taken place in the humero-ulnar and superior radio-ulnar joints, but this is due to extension from the radio-humeral



segment in a simple and mechanical manner. Fig. 1 represents diagrammatically the changes which such a joint presents. The part is looked at from the front.

You may remember that at an earlier lecture we found the conditions which this specimen presents of very considerable service indeed when we were considering the manner in which force was transmitted directly from the radius to the ulna, together with the function of the ulna and of the inter-osseous membrane, and the share these structures took in the indirect transmission of force in the same direction to the humerus. It is most unfortunate for the student that anatomical works are arranged on a principle which is utterly incomprehensible to me. It would seem that, in proportion as a structure of the body is of no obvious practical importance, in that proportion does the anatomist spend time and energy in its description. On the other hand, if a part is of

infinite practical interest to the surgeon, the anatomist regards it with so much contempt that he limits his description of it to a few lines, which are but too frequently incomprehensibly obscure and meaningless, if not thoroughly incorrect. Take, for instance, the description of the movements of the elbow-joint in p. 172 of *Quain's Anatomy*, vol. ii., part ii. What can the student learn from it as to the physiology and mechanics of the elbow-joint, and is the inter-osseous membrane such an unimportant structure that its function requires no description? Yet this is the best work on Anatomy in the English language.

If, however, the student wishes to learn about the lobes and fissures of the cerebellum, the same work describes them in such detail as to produce in some minds a sense of over-repletion, combined with one of wonder as to the particular advantages to be derived from this knowledge in the practice of one's profession. One longs for a little more perspective in the teaching of anatomy.

2. A man, passed middle age, falls heavily upon his side, striking the great trochanter with much force. In consequence of this injury he experiences much local pain in the hip-joint, which is aggravated by movement of it. If the legs be measured, they are found to be of equal length. The patient finds that after the injury his leg is not so reliable as it was before—that the movement of complete flexion of the hip-joint is accompanied with discomfort at first, and later with pain, so much so that any movement of the joint is avoided as much as possible. He also suffers discomfort or pain in the joint in damp weather, or during the cold east winds. I believe that it is the similarity of this pain to that felt by rheumatic patients that has led clinical observers to include this traumatic condition among those classified under the old headings of "rheumatoid arthritis," etc. I need hardly point out that after an injury to a bone, such as fracture, etc., the sufferer often complains of the same kind of pain under the same atmospheric conditions. After a time the patient notices that the damaged leg has become shorter than its fellow, and actual measurement shows that this is the case. This shortening increases during the life-time of the patient, if he continues to throw his weight upon the articulation. The joint becomes progressively less secure until at length the weight of the body cannot be

supported by it at all in many cases. Soon after the receipt of the injury grating can be felt on flexing or extending the joint, and this sensation becomes more marked as time goes on. If such a joint be examined after death, the head of the femur and the acetabulum are found to be completely altered in form and character. The opposing surfaces of the innominate bone and

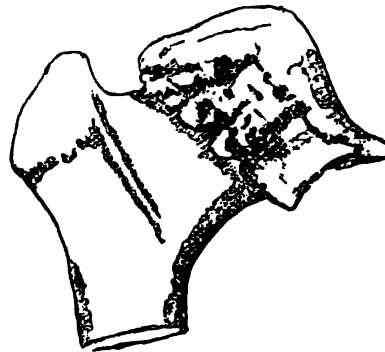


Fig. 2.

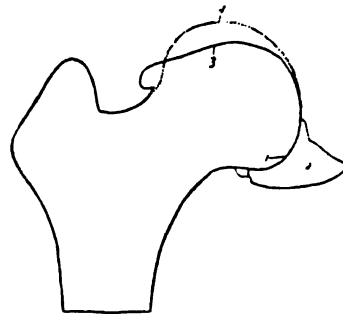
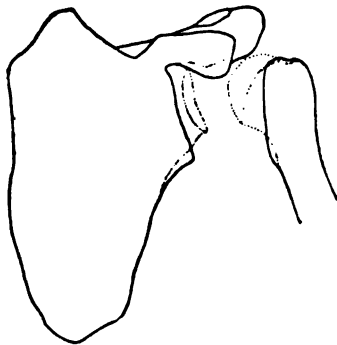


Fig. 3.

femur are rubbed down, sclerosed, and eburnated, while around these eburnated surfaces there is an abundant deposit of more or less dense bone, whose obvious function is to compensate for the altering direction in which force is transmitted through the joint, and to render it as secure as possible under the circumstances. The capsule has become thin and lax, and has ceased to perform most of its normal functions. The synovial membrane is bulky in the joint, and contains an abnormal amount of synovia. The causation of the several changes here described is quite obvious. Figs. 2 and 3 represents the condition and its mode of production from the normal state. The opposing surfaces of articular cartilage have been bruised sufficiently to interfere with their

vitality, and there being no means of replacing the damaged tissue it is removed. The habitual transmission of force through the damaged joint results in the progressive destruction of the remainder of the articular cartilage, in the exposure, eburnation, and the progressive gradual destruction of the subjacent articular lamella of bone. These changes are followed by the others already described, the factors determining their evolution being solely mechanical. This all seems very simple and obvious to you. Yet at the time I wrote the paper already referred to in the Transactions of the Pathological Society, this condition was, and, for all I know, may still be described in a well-known text-book of surgery as "interstitial absorption of the neck of the femur." Such a pathology is founded on imagination and not on observation.

3. The following serves as another instance of chronic traumatic arthritis. A man falls heavily on the outer surface of his shoulder. He suffers from much pain at the time, and he can neither bear the shoulder-joint to be moved, nor can he perform any active movement himself, on account of the pain associated with it. If the shoulder be examined no fracture can be made out, and the



outlines of the humerus and of the glenoid cavity are normal. The symptoms progress as in the case of the hip. The shoulder becoming flattened, the patient is unable to abduct the humerus, and the movements of the shoulder-joint become limited finally to a moderate amount of flexion and extension. On examining the joint after death, the opposing surfaces of the head of the humerus and of the glenoid cavity are found to be deprived of

cartilage, the articular layer of bone is rubbed down and eburnated, and the surface of the humerus is rendered flat or but slightly convex, being much increased in breadth by the deposit of bone upon its margin. The articular surface of the scapula has undergone similar changes. The synovial membrane is bulky, and the muscles of the shoulder, especially the abductor muscles, are much wasted. Here, again, the causation is quite obvious. Force is applied to the outer surface of the humerus in such a direction that its articular surface is driven forcibly against that of the scapula, and in a direction at right angles to the surface of the latter. Changes ensue in the manner already described in the hip.

4. I would instance still one more example in the same joint. An elderly man receives a heavy blow, or pitches heavily on the point of his shoulder, breaking his acromion process, whose loose fragment is driven forcibly downwards upon the upper surface of the adducted humerus, bruising its articular surface severely, together with the intervening capsule and tendon of the supra-spinatus. Degenerative changes ensue in the contused portion of the articular cartilage, and gradually extend to more or less of the remainder of the cartilage of the head of the humerus, and to that of the glenoid cavity. The upper portion of the capsule with more or less of the tendon of the supra-spinatus is gradually destroyed, and the humerus comes sooner or later into direct contact with the movable fragment of the acromion. The constant movement of these bones upon one another results in further changes in both, the acromial portion becoming excavated, eburnated, and lipped. The intra-capsular portion of the biceps tendons is destroyed, the long head acquiring an attachment to the altered great tuberosity. Other changes develop at the same time in the synovial membrane. I would call particular attention to this last injury, as it bears in an important manner on the elucidation of the causation and pathology of chronic traumatic arthritis. I showed you in an earlier lecture that fracture of the acromion is of very common occurrence, and that the supposed rarity of the fracture depends upon a want of skill in recognizing its existence. Also, that union is due to the fact that unless the accident is diagnosed, which it very rarely is, or unless it be accompanied by some more severe injury as fracture of the upper end of

the humerus, the shoulder is not kept at rest for a sufficient time to allow of the acromial fragments uniting by bone, and as a consequence a false joint forms. When the blow which produced the fracture was sufficiently severe to contuse the articular cartilage of the head of the humerus, and the intervening soft parts, the changes I have just described follow.

Anatomists are largely responsible for the non-recognition of these fractures, since they have chosen to regard the resulting ununited conditions as examples of non-union of an epiphysis, and, so far as I can see, upon no reliable evidence whatever. Some surgeons have readily accepted the statements of the anatomists, apparently without investigating them. They have, however, observed the frequent association of this so-called ununited epiphysis with the presence of so-called rheumatoid changes in the shoulder-joint, and have explained the cause of their association in various ways. One has gone so far as to assert that in cases of chronic rheumatoid arthritis of the shoulder-joint, a disunion of the acromial epiphysis from the rest of the bone takes place. Such a statement hardly calls for criticism, and I only mention it to indicate the frequency with which those changes which the pathologist regards as characteristic of the disease, rheumatoid arthritis of the shoulder-joint, are found associated with ununited fracture of the acromion. Can anything be more obvious and simple than the mode of development of an ununited fracture of this process of the bone, if one considers for one moment its position and muscular attachments, together with the difficulty of retaining the broken fragments at rest for any length of time, even if the fracture be recognised? The only two examples of bony union which I have observed followed a fracture of the acromion, which was accompanied by a severe comminuted fracture of the upper end of the humerus. Owing to the pain resulting from the presence of the latter fracture, the shoulder was kept at rest sufficiently to allow of bony union. Though I might multiply such instances indefinitely, I think I have said enough to show that a severe injury to a joint is liable to be followed by the development of such pathological conditions as surgeons are still accustomed to regard as evidence of the presence of rheumatoid arthritis.

ABDOMINAL SECTION: GENERAL OBSERVATIONS ON DETAILS AND AFTER-TREATMENT.

BY

ALBAN DORAN, F.R.C.S.,

Surgeon to the Samaritan Free Hospital.

Two years ago I contributed to this Journal a few notes on the feeding of patients after abdominal section.* In conclusion I observed that full attention to after-treatment is one of the best ways to attain successful results. I admitted that it is at first less interesting to contemplate than the question of antiseptics, short incision and drainage, but it is quite as important.

Further experience has not led me to modify my practice in respect to diet and the management of the patient's bowels. I believe rather more than I did in 1894 in the value of nutrient enemata as compared with starving, or early feeding by the mouth. They counteract shock, and nourish without causing flatulence. No irritation of the bowel need be feared if the most ordinary precautions be taken. Later on, if symptoms of obstruction or peritonitis set in, these enemata are of yet higher value. I find that, if mixed with a little lime-water, milk is not so objectionable an article of diet as I once thought it to be. A few ounces daily, after the second day, aid greatly in keeping up nutrition, especially in old women, and if not given pure or in excess, the fear of the development of scybala is not great. A dose of sulphate of soda, citrate of potash, and glycerine dissolved in water may be given two or three times daily from the fourth to the sixth day in constipated subjects, or in cases where the passage of solid motions might be dreaded for special reasons. About twenty grains of the sulphate of soda are sufficient as a rule. The citrate must not be given unless the urine is concentrated and charged with urates, or that excretion will become alkaline. After two days' treatment with this draught, an enema will bring away a semi-liquid motion or soft solid fæces without trouble.

I still find the enema greatly preferable to pills

* "On the Feeding of Patients after Abdominal Section," Vol. III., 1893-4, p. 346.

and aperients in simple cases, and also in very bad cases, where any extra irritation of the stomach and upper part of the intestinal tract should be avoided. On the other hand, I continue to give aperients when the patient is evidently suffering from the constipation alone, or from the diet disagreeing with her. Headache, furred tongue, and distinct loss of appetite, with no grave abdominal symptoms, require treatment beginning in the upper part of the alimentary canal.

Diet is one of the most important of the numerous matters of detail on which the patient's life and comfort depend. There remain many others, some of which may be profitably discussed.

The length of the abdominal incision is a matter of some importance, and I find that the practice of different surgeons varies in this respect. Before Sir Spencer Wells succeeded in establishing ovariectomy as a justifiable operation, long wounds were made, and the tumour was hastily pulled out. He showed how a cyst could be tapped through a short incision, and extracted at leisure afterwards. The clamp did not necessitate enlargement of the wound. When the ligature came into vogue, it was found that the pedicle could, as a rule, be easily and safely secured through a short wound. So far experience favours the short incision. I seldom make the wound longer than three inches in the case of a non-adherent cyst. Many large tumours can be extracted through a two and a half inch incision, and the pedicle secured without any bruising of the wound. I have never had reason to regret this practice.

Unfortunately, this is an age of record-beating, and one or two surgeons have boasted too much of their short incisions. When deep manipulations are required, or when the tumour is firm, the wound may get badly bruised by the surgeon's hands, or by the pressure of forceps, or by the tumour during its extraction. In operations where the abdomen is not distended, the incision should be made at least three inches long at once. Otherwise the recti may become severely bruised. When suppurating tubes or dermoids are removed, a bruised wound is particularly liable to suppurate.

The surgeon must never be ashamed of enlarging the wound if necessary. I feel sure that one reason why a wound is often made long from the first is the fear that it "looks bad" to make the incision longer during operation. This is a

most unsurgical principle. There is but one sound rule concerning the abdominal incision. It should be made as long as appears necessary, and if, during the course of an operation, it is found to be too short to allow of manipulations without damage to its edges, it must be made larger. Long incisions are specially needed in cases where obstructed or diseased intestine requires to be searched for and examined.

The suture question remains unsettled. I prefer silkworm gut to silk. If properly softened in water before the operation, and not tied too tightly, the gut will not cut through the tissues of the abdominal wound. In fat subjects, especially after hysterectomy and application of the *serre nœud*, I find that it is best to suture the peritoneum separately. In other cases, a single set of sutures passing through all the layers answers perfectly. I have examined the cicatrices of abdominal incisions from two to ten or eleven years after I had made them, and found them perfectly strong. Novices often wonder why some of the stitch tracks suppurate, as they have taken every precaution to render the sutures aseptic. The truth is that the inexperienced hand is often rough, and the suppuration is due to the passage of sutures through tissues bruised by the hands or by instruments.

It is certain that the peritoneum must not be transfixd by the suture-needle too far from its cut edge, especially when all the layers of the wound are included in the suture. If over one-eighth of an inch be taken upon each side, two broad surfaces of serous membrane will project forward lying in apposition between the more superficial layers. This will effectually prevent the union of those layers. In a thin wound the edges of the serous membrane may, after the tying of the sutures, lie level with the surface of the abdomen, if too much peritoneum be taken up by the needle. I observed this condition once in one of my own operations, and immediately withdrew the one suture which had been entered too far from the edge of the peritoneum, and passed it in again nearer to the edge. It is evident that if this mistake be not rectified a hernial pouch will most certainly develop. Hernia of the cicatrix is an intolerable nuisance to the patient, and may cause her more trouble than did the successfully removed tumour. Believing no longer in surgery,

she is apt to delay till the hernia becomes very large, and full of diverticula. I have had to operate for the radical cure of a hernia of this kind. The operation is always difficult, and never free from danger. Omentum and intestine usually adhere, more or less firmly, to the serous lining of the diverticula or, worse still, to the fibrous neck of the sac. It must be remembered that this ugly complication is often due not to neglect on the part of the operator, but to the patient's carelessness about wearing the belt after convalescence.

As it is so important to avoid hernia, there are precautions which it is advisable to take when the sutures are removed. The precise date can only be determined by common-sense surgical principles. I find that when the incision is properly dressed at the operation it may be left alone for a week. Then half the sutures should be removed. When a very large tumour has been taken away the parietes will still be found flaccid, and the strain on the edges of the wound will be trifling; though the operator must beware of flatulent distension. In the contrary condition, where there was little previous distension, the muscular layers remaining strong and unstretched, the strain on the wound will be considerable. In any case the abdominal walls should be well supported by strapping and bandaging after the removal of the sutures. On the ninth or tenth day the remaining sutures should be taken away. An assistant must press with his hands on the parietes during this dressing, and continue to do so till the strapping has been applied. Then the abdomen is once more bandaged. In this manner the new cicatrix is protected from all straining. The surgeon must not forget, however, that flatulent distension is a powerful mechanical agent in stretching the wound. For this reason an abdominal binder should be put on by the patient at night for several months after she has worn the belt during the daytime; besides, all causes of flatulence must be avoided.

I find that it is advisable to use strapping from the first, since it is more reliable than the bandage and padding alone. At first, it protects the walls against the evil effects of chloroform-sickness, and then, as well as later, it supports the wound, promoting firm cicatrization. Loose bandaging and inefficient strapping allow of flatulent distension,

a source of grave peril during the early days after operation, and, later on, a cause of the awkward complication already noted.

The temperature is a serious matter. Here there has been too much record-beating, and I have heard surgeons boast that they "never have temperatures." An honoured authority goes so far as to say: "If I had to deal with a temperature above 104° lasting for more than two hours, I should administer a full dose of antipyrin, and possibly supplement it with sponging of the cutaneous surface." * This passage in a well-known work is, in the experience of many surgeons, open to grave criticism. A temperature above 104° , lasting for more than two hours, represents conditions not improved by antipyrin. The much-abused ice-cap is far more comforting to the patient than sponging. If properly adjusted it not only makes the temperature fall, but often promotes sleep after distressing restlessness. The surgeon must never forget that when he cannot remove the source of the high temperature, the high temperature is all the more an evil in itself.

A rise of temperature with but little quickening of the pulse and no distension, retention of flatus or vomiting, often means, during the first few days, the coming on of menstruation, especially when the catamenia appear before they are due. The same symptom in the second or third week usually implies the presence of scybala retained in the large intestine. Low temperature and high pulse are commonly associated when intestinal obstruction from purely mechanical causes is present. Sepsis in any form involves more or less rise of temperature. This fact is the true reason why high temperatures are dreaded, and why men boast that they have "no temperatures." Every surgeon must avoid that evil state of mind which tempts him to believe and to state that other men lose their patients from sepsis, whilst his own scanty mortality is due to unavoidable and purely accidental causes.

A rapid pulse without other bad symptoms is occasionally seen, and is sometimes a neurosis. In one instance in my practice, where the patient was an anæmic girl, it simulated hæmorrhage, of which, however, no other signs were present, and on the second day the pulse was normal. A cyst con-

* J. Greig Smith, "Abdominal Surgery."

taining much fluid had been removed, but rapid pulses do not necessarily follow the removal of large tumours. In a case of hysterectomy the pulse became alarmingly frequent. I had used iodoform freely as a dressing for the stump of the pedicle. Directly I discarded that compound the pulse fell, and gave me no further anxiety.

What I presume to be a true neurotic pulse may be noted when a patient is in dread of the dressing of the abdominal wound, or is worried or excited by relatives. The rise of pulse or temperature after a visit from friends may mean more than a neurosis. The patient may have moved about, or taken improper food, and thus set up local mischief.

In conclusion, the patient's life and comfort depend on a scrupulous attention to matters of detail at and after abdominal section. These matters must be clearly understood by the operator. He must not make too little or too much of cardinal symptoms; above all, he must not look upon them as a special credit or disgrace to himself. They are guides to the patient's condition. To save the patient and prevent sequelæ is not sufficient. The surgeon must do his utmost to make her as comfortable as possible during the critical two or three weeks which follow the operation which he has undertaken.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Reported by J. T. CONNER, M.D.

Two Cases of Kaposi's Disease (Xeroderma Pigmentosum).

THE first was a man, æt. 21, brought by Mr. J. Startin. Since he was two years old he was liable to blisters. His face was freckled, and stigmata were also present. The skin here, as well as on the neck, was tight, and ulcerated in places. There was a general condition of xeroderma (congenital). On the palms the skin was dry and thick. He had lost all his nails. The

legs were red and inflamed. There were symmetrical bullæ on the knees and shins. No freckles could be seen on the arms or legs. He stated that when his skin was rubbed, blisters were produced easily. The teeth were broken and decayed. His brother was much freckled, and his sister a little.

The second was a girl, æt. 8, sent from St. Bartholomew's Hospital by Mr. Willett. She had been freckled since she was two years old. The face and neck were thickly covered (much more so than in the first case). Here and there were small ulcers, with brownish crusts. On the arms the pigment shots reached as high as the elbows, and the palms were affected as well as the backs of the hands. The skin of the legs was morbidly dry. A few freckles were to be seen on the backs of the legs, reaching as high as the popliteal spaces. There was no general condition of xeroderma, and the nails were good. The conjunctivæ on the lids was red and inflamed.

Remarks.—Mr. Hutchinson thus explained the pathogenesis of this complaint. It is a "family disease"; that is to say, it affects certain of the children born of healthy parents, while others are exempt. Retinitis pigmentosa belongs to the same class. In the one case the subject is born with a skin, and in the other with a retina, which "does not wear well"—it is unduly sensitive to light.

The reaction produces an abnormally pigmented skin or retina, as the case may be. But he had never known the two conditions to exist in the same patient. The freckles first affect the parts exposed to light—face, arms, and legs. But the process is an aggressive one; so they spread to unexposed parts—upper arms, thighs, and clavicular regions. Also the irritation leads not only to pigmentation but to inflammation. The vessels dilate, stigmata are formed. Ulceration of a lupoid type occurs. Like lupus, this ulceration in its turn produces cancer.

He objected to the name of xeroderma pigmentosum, as xeroderma was usually absent, and when present only a coincidence.

Prurigo Hyemalis.

A man, æt. 26, sent by Mr. Waren Tay, had suffered from attacks of this disease, in winter.

since 1888. In summer time he was exempt. He was severely attacked from 1888 to 1891, and attended Blackfriars Hospital under Mr. Hutchinson. From 1891 to 1895 he was much better. Now he had a very severe attack. The eruption was general, and distributed chiefly, but not exclusively, on the regions covered by the vest, the loins being much the worse. But the centre of the abdomen and back were almost exempt. There was very little on the legs; the hands, wrist, and penis were not attacked. It consisted of hard papules discrete in general, but becoming confluent in parts, and here and there pustular. He did not associate the eruption with any change from a cotton to a woollen vest.

Remarks.—In these cases the skin becomes abnormally irritable under the influence of cold. This leads to scratching, which in its turn produces the eruption. The exemption of the penis and wrists distinguishes it from scabies, and the fact that it does not exist from infancy from Hebra's prurigo. In some cases the skin never gets quite well, but is always liable to itch.

Multiple Sarcomata of the Subcutaneous Tissue.

A woman, æt. 52, sent by Dr. Dowding, of Wanstead, had a growth about three inches in diameter at the left margin of the sternum, and between the second and third ribs, adherent to the bones, but not to the skin. It was of a year's duration. Below the left axillary region were three small nodules, about the size of hazel nuts, which were freely movable in the subcutaneous cellular tissue. They had been noticed for three months. Between these and the primary growth were a number of still smaller ones. The tumours were very tender, but otherwise not painful. She had lost flesh considerably.

Remarks by Mr. Hutchinson.—The secondary growths are at a distance from any lymphatic glands, and are probably the result of deposit in the lymphatic vessels leading from the primary growth.

Psoriasis of the Nails.

A young man, æt. 30, showed this condition in all the nails of both fingers and toes. The nails were grey, opaque, and thickened. But the sur-

face was smooth, and the lunulæ sound. The skin was normal. He had suffered from syphilis two or three years ago.

Remarks.—Mr. Hutchinson said this was the most typical case he had seen of psoriasis of the nails. The disease is produced by accumulation of dead epidermis below the nail, whilst the free surface and root remain unattacked. This produces opacity and loosening. Though the result of syphilis in this case, the condition was entirely different from that usually produced in the nails by this disease.

Relapsing Herpes of the Tongue and Mouth, the Result of Syphilis.

A lady, æt. 51, brought by Mr. G. W. Sequeira, had large, inflamed, abraded patches, covered with a thin pellicle near the tip of the tongue, both on its upper and under surfaces. The tongue was bald from loss of filiform papillæ. On the floor of the mouth, and on the mucous membrane of the cheek, were also patches covered with a pellicle. The present attack had lasted four months, and she had been liable to similar attacks for 20 years. There was a history of syphilis previously.

Remarks by Mr. Hutchinson.—The position of the inflamed patches, at the tip and margins of the tongue, and the relapsing attacks, are characteristic of herpes. It differs from the "smoker's tongue" (leukoplakia), in that the patches are more inflamed, and not covered with such a thick membrane.

Paresis of the Internal Recti and Double Ptosis in a Boy.

A boy, æt. 13, sent by Mr. Waren Tay, was suddenly affected three weeks ago with double vision, divergent squint, and inability to raise the eyelids fully. These symptoms have remained stationary. He can fix with either eye, but usually uses the right, whilst the left is deviated outwards slightly. But this symptom is sometimes absent. When he looked to the left the right eye did not completely turn inwards. There was, therefore, some weakness of the right internal rectus, as well as the left. The upper lids partially covered the globes, and he held his chin up and wrinkled his brows to compensate this. The movements of the iris and the accommodation were normal. He

had frontal headache at times. This preceded the onset by a week. One week before this he had slight sore throat. The soft palate moves freely, and the knee-jerks are normal. There are no indications of hereditary syphilis.

REVIEWS.

Medical Electricity. By LEWIS JONES. Second Edition, 1895. (H. K. Lewis.)

Price 10s. 6d.

That a second edition of this work should be called for in such a short time after its first appearance, is proof enough that it has found a large number of readers who appreciate it; nor is this to be wondered at, for certainly we know of no other work which gives with such clearness the dry, but necessary, details of electricity in its application to medicine.

To know what can and what cannot be done by electricity, and how to do that which is possible, is, we fear, the possession of too few of the members of our profession, but we can assure our readers that the knowledge is to be fairly easily (it never can be very easy) and pleasantly acquired by a perusal of Dr. Jones's book. We congratulate both author and publisher on the appearance of this edition, and trust that a third will soon be required.

Injuries and Diseases of the Genital and Urinary Organs. By HENRY MORRIS. (Cassell & Co.)

Quoting from Mr. Morris' Preface, he says, "I have endeavoured to make my descriptions as clear and complete, but, at the same time, as brief as possible." We can say that this endeavour has been most successfully carried out. From the rarest accident to the simplest disease of the parts dealt with, we can scarcely find an omission of any importance; the language in which they are described, too, is vigorous and plain, and unencumbered by any unnecessary details of pathology, while the essential points are given with great distinctness. The book is beautifully illustrated

by 96 woodcuts, all of excellent quality and clear in detail. Chapter IV., on functional disorders in relation to the testicles, we think is worthy of special notice for the excellent advice on the treatment of masturbation, impotency, and sexual hypochondriasis. We have recently expressed an opinion on the propriety of a man writing a book, or, rather, publishing one, while still exercising his function as an examiner; but, as the work before us is hardly a student's book, the objection has here no great weight. To busy practitioners who desire a work of reference we can most heartily recommend the book, which is sure to prove attractive from its style and completeness.

The Year Book of Treatment, 1896. (Cassell & Co.)

Price 7s. 6d.

The burden of new drugs, new diseases, new instruments, grows heavier every year, and melancholia increases in proportion; it is apparently useless to protest, and so we must go on taking the bushels of chaff for the sake of the single corns. Messrs. Cassell, by their editors, have certainly tried their best to do the winnowing work, and the names of Sidney Coupland, Ralfe, Hale-White, Garrod, Edmund Owen, Herman, P. Manson, amongst others, are a guarantee that what is here preserved is worth at any rate extended trial. Certainly nothing seems to have been omitted that promises good results, and we can advise the busy practitioner that he has here a rich mine of easily gathered nuggets of therapeutics.

Early Scoliosis. By PERCY G. LEWIS. (Ball & Sons).

This little book of 49 pages contains all that need be known of the treatment of early spinal curvature; its principles are sound, and the treatment admirably adapted to the ends in view, while it is so simple that anyone can most readily apply it. We may sum it up in the author's words: "The practice of gymnastics, *properly carried out*, is the natural cure of scoliosis." If this little book were better known, we should hear much less of spinal supports, jackets for curvature, and all such abominations; and we trust it will be read by everyone who has children to deal with. It is well illustrated by woodcuts.

THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 19, 1896.

CLINICAL LECTURE

ON

TWO CASES OF ULCERATIVE ENDOCARDITIS WITH EMBOLISM.

Delivered at the London Hospital, Feb. 11, 1896,

BY

FRANCIS WARNER, M.D. Lond., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—The subject on which I propose to speak to-day is ulcerative endocarditis producing embolism. Two cases have recently been under my care in the wards, which illustrate the diagnosis and course of this very fatal disease.

The first case is that of a woman, æt. 22, who was engaged in domestic service till the commencement of her illness. She came to the hospital as an out-patient, complaining that for the last three weeks she had suffered much from pain in the chest and left side, with attacks of shortness of breath. The pain had commenced somewhat suddenly one night when she was going to bed; for a time it got better, but had returned on several occasions. On cross-questioning her, it seemed that for about three or four months, although able to continue her work, she had suffered from breathlessness, and occasionally noticed slight œdema of the legs. When admitted to the wards, it was ascertained that the history of the family was generally healthy, and there was an absence of rheumatism, except that the father was said to have had slight rheumatism occasionally. This young woman had previously been in the hospital, as an in-patient in September, 1894, for rheumatic fever, but she was discharged apparently in good health, and had not suffered from cardiac symptoms until the date stated. She was a thin, pale-looking woman, but when seen in bed she said the chest pain had entirely subsided. The lungs were clear, the urine somewhat scanty, measurement showing it to be 22 ozs. per diem on

the average; specific gravity 1022, with a deposit of urates, but no albumen. The bowels were costive, tongue red and slightly furred in the centre. On physical examination the pulse was found to be small—80 per minute—the apex beat was displaced downwards and outwards, external to the nipple line. There was a distinct and well-marked systolic bellows mitral murmur, well conducted to the axilla. The apex beat was forcible, indicating the degree of hypertrophy of the left ventricle. There was also a soft systolic murmur heard at the second cartilage, slightly conducted upwards, but no regurgitant diastolic murmur was heard. There was also a soft systolic murmur over the pulmonary valves; and it appeared probable that both these basic murmurs were due rather to anæmia than to the organic condition of the semilunar valves. The temperature was 100° on the day of admission, but, as we shall see, it was often above this in the course of the disease. It is remarkable that often the temperature was subnormal, sometimes at night, sometimes in the morning. The weight of the patient on admission was 6 st. 12 lbs. During the first week she visibly lost flesh, and her weight diminished by 1½ lbs.

In the second week the patient began to complain much of head-pain, which was severe; also of insomnia, so that she experienced considerable difficulty in getting any sleep, even by the use of hypnotics. During the second week the average temperature was 100°, but often it was subnormal. On November 21st—nine days after admission—the patient suffered in the morning from severe headache; then she suddenly complained of severe abdominal pain, pointing to the region of the spleen as the site. The spleen was plainly enlarged, and extended 1½ inches below the ribs; it was tender on palpation. There appeared every reason to think that the pain was caused by sudden embolism occurring in the spleen, and this was confirmed post mortem. At five o'clock on the same day she was noticed to be in some sort of fit, or suffering from some sort of cerebral attack. The breathing was stertorous, the limbs

motionless, the corneæ appeared quite anæsthetic, and gave no reaction in movement. The pupils were small, but later on were dilated. At the same time there was found to be some rigidity of the left arm and leg, and as soon as she recovered she complained of pain about the left hand, indefinitely localized. The knee-jerks were found to be absent, and fifteen minutes after the attack commenced there was ankle-clonus on both sides. The urine was passed unconsciously. The pulse at the wrist was unequal on the two sides, being markedly weaker on the left than the right, and so it remained. It seemed very probable that on this day a second embolism had occurred, partially blocking the brachial artery or some of its branches. The temperature at this time fell to subnormal, viz., 97° F. A little later in the day there was noticed to be conjugate deviation of the eyes to the right, both eyes being fixed in that direction without squint; but this only persisted for an hour. The importance of this latter sign would appear to be that it pointed to some irritative cerebral lesion on the left side at the same time. I think it is probable that her head pain, which was severe, was also an indication of disturbed circulation in the brain. The next day, November 22nd, the head pain was still severe, the patient was restless and looked ill, the knee-jerks were still absent. The face had a vacant appearance, and facial expression was, to some extent, lost; in fact, the cerebral aspect of the case was marked. The urine was now 1022, with a deposit of urates; there was also retention of urine—another indication of cerebral disturbance. On November 26th the knee-jerks were still absent, the headache severe and without remission; urine denser and still scanty—sp. gr. 1036, with deposit of phosphates. The next day, the 27th, she was slightly better, the headache was somewhat less, and now that she could speak more she complained of pain in the left arm, just above the site of the elbow. This provided a further indication that probably the embol in that limb was in the brachial artery. The left radial pulse was still weak, and the temperature higher during the next few days, being between 100° and 102°, but still frequently fell below the normal. There was no inflammation in the throat or elsewhere in the body to account for the temperature, so that it seemed clearly due to the cardiac and cerebral

condition. Retention of urine continued, and the enlarged spleen could be felt; the bruits remained as described on admission; and I may say that there was no change in the physical signs during the progress of the disease. On December 2nd the spleen was very distinctly enlarged, it could be felt one inch below the ribs, and was tender; the patient looked very exhausted, pale, and emaciated. She spoke in a very low voice, and complained much of pain in the abdomen and in the head. The apex beat was $1\frac{1}{2}$ inches below the nipple.

Thus the case went on, and on December 16th the head pain was more severe than ever. The patient vomited several times; this appeared to be due to the cerebral disturbance. The highest temperature at this stage was 102.5°, and now albumen was found in the urine. This appeared to be due to other emboli having occurred, causing collateral fluxion and hyperæmia in the kidney. The tongue became dry, she vomited several times, could obtain very little sleep, complained of pain in the back, palpitation, and shortness of breath. She looked extremely pale and exhausted, and was evidently very ill; the pulse was 112, and the general loss of power and loss of voice were more marked than ever. Ophthalmoscopic examination, which was made on this and several other days, showed the retinal appearances to be normal. On January 11th, purpura appeared on the back, she could scarcely swallow, sordes formed about the lips, and the voice was very feeble. The splenic enlargement had nearly subsided, the pulse became very weak, but the lungs remained clear. The breathing became stertorous, and the patient died on January 14th.

Regarding treatment, the drugs which were useful we can only speak of as palliatives. Sulphonal; pulvis ipecac. co., purgatives and sedatives; a mixture containing bromide of potassium and hydrocyanic acid was given to quiet the patient, and check the vomiting. During the greater part of the illness the patient took digitalis, quinine, and salicylate of soda.

At the post mortem examination, a considerable bed sore was found over the sacrum and hips, which had been forming for the past fortnight. On section, the left pleura was adherent to the walls of the chest and also the pericardium. The lungs were cedematous and congested, and the pericardium was adherent both to the pleura ex-

ternally and the heart internally. The heart showed considerable hypertrophy, especially in the left ventricle; its weight was 26 ounces. The heart muscle was firm, and seemed to be the seat of true hypertrophy. In the left auricle, at the base of the mitral valve and extending upwards $1\frac{1}{2}$ inches, also at the margins of the valve, it will be seen in the specimen on the table, that there is a mass of fine prolific fungating granulations; the edge of the mitral valve is found to be crowded with profuse small granulations continuous with the auricle, many of which can be readily detached. The successive emboli indicated by the history occurred, first in the spleen, probably next in the brain, then in the left brachial artery, and others seem to have found their way to the kidneys. The aortic and pulmonary valves were healthy. The spleen, which I also have here for your inspection, is considerably enlarged, and weighs 14 ozs. Two considerable infarcts, about the size of walnuts, V-shaped, and of a yellowish colour, with the bases towards the surface of the organ, can be seen, as well as two smaller ones. The kidneys were congested, the two together weighing 11 ozs. but no visible infarcts were demonstrated in them. The liver weighed 3 lbs. 12 ozs., was friable, and somewhat congested. The brain was anæmic, but no coarse changes were visible to the naked eye.

The second case was that of a man, æt. 35, who had, all his working life, been employed in gas works, his labour being laborious. He was first seen at the hospital on December 10th, when he presented himself at the out-patient department, complaining of shortness of breath, cough, and expectoration, and showed signs of considerable dyspnoea and distress. He was at once admitted to the wards. The family history showed no tendency to disease. His father and mother were both alive and well; he had a brother and three sisters alive and in good health. One brother was said to have died of "dropsy" at 7 years of age, but there was no proof that he had had rheumatism, or that his dropsy was due to heart disease. The patient had rheumatic fever fourteen years previously, and it appears to have been a severe attack, as he stated he was laid up for thirteen weeks. He eventually recovered his usual health, having experienced no heart symptoms as a result of that illness, and he

worked until about three weeks prior to his admission to hospital. He was not aware of anything the matter with him until four months before admission. At that date—August, 1895—he began to notice a sensation of tightness across the chest, and this was followed by cough, and expectoration of greenish-black phlegm. This, however, did not compel him to cease work until a few days before he sought advice, when he was suddenly seized with pain in the head, which continued for two days, and then as suddenly disappeared. The man's condition gradually became worse, his head pain and distressful breathing persisting until he came into the ward on December 10th. When seen the second day after admission, he was lying quietly on his back and said he was quite comfortable, all his distress having left him. His face was pale and anæmic, and the tips of his fingers somewhat blue; temperature 100° . During the second week the temperature rose to 102° , and remained at about that height until nearly the end of life, with morning remissions. In fact you will see the temperature chart bears a considerable resemblance to that of typhoid fever, and cases of ulcerative endocarditis have been mistaken for that disease. The urine was normal; no albumen; sp. gr. 1030, with deposit of lithiates. Digestion, nothing worth recording.

As to the cardiac signs, the pulse was 90, full and collapsing. Palpation of the heart showed considerable largeness and hypertrophy, the impulse being forcible. The apex beat was $1\frac{1}{2}$ inches below and a little external to the nipple line. A distinct thrill was felt here on palpation. There was a loud mitral systolic murmur, conducted to the axilla, also a presystolic murmur at the site of the apex beat. At the base, a systolic murmur was heard without any regurgitant murmur. The breathing at this time seemed laboured; coarse râles were heard over the left base behind. He still had cough, and continued to expectorate greenish-black mucus.

The patient improved considerably, and was able to be up for a time. On December 18th the patient suddenly complained after dinner of pain in the left leg and foot. No pulse could now be felt in the left dorsalis pedis artery, and it was evident that an embol had blocked it. On the 23rd the pain had gradually disappeared, but the

arterial pulse remained absent, and so till the end. The patient, however, felt better, and the pain had subsided from the leg; in fact, he was so much better that he was put on middle diet.

On January 2nd the man complained of fulness and pain at the pit of the stomach after his food, the food seeming to lie heavy on his stomach; he also complained of flatulence, and vomited. Later in the day he seemed to be very ill; profuse perspiration occurred, and he was quite unable to sleep, even with the aid of sulphonal and hydrate of chloral. Stimulants were used, but his condition became gradually worse, and he died on January 4th.

At the post mortem there was found to be considerable hypertrophy of the heart, which weighed $27\frac{1}{2}$ ozs., and may best be described as a large bovine heart, with considerable thickening of the walls of the left ventricle, the hypertrophy being sound. The pulmonary and tricuspid valves were normal; the mitral valve admitted two fingers. On the anterior curtain of the mitral, just below the left posterior aortic flap, was a fungated mass of beady vegetations, about $\frac{3}{4}$ inch in diameter, irregular in outline, and projecting forward about half an inch from the surface of the endocardium into the cavity.

The vegetations composing the mass were very small, and easily detached on manipulation. The aortic valve was also affected with old organic deformity and thickening of the valves. The anterior and right posterior edges were diffused into one; the left was the more deformed. From its margin there projected an irregular fungating mass nearly half an inch in diameter, and complete ulceration had occurred at the site of this valve. There were no pleural adhesions. The lungs were tough, with an increase of fibrous tissue. The spleen weighed 5 ozs., and the capsule was somewhat thickened. The kidneys weighed 14 ozs., and were somewhat granular upon the surface. Some infarcts were found in both kidneys. In the right kidney was an old fibrous patch, indicating the site of a former infarct.

Both the cases which I have related to you present well-marked examples of so-called ulcerative endocarditis, and in many particulars their clinical history and the symptoms of the disease agree. In both cases there was old-standing valvular disease, probably of many years duration, as indi-

cated by the considerable degree of hypertrophy, probably dating from rheumatic fever. So that after the endocardium had become first damaged there had been a considerable interval before the outbreak of the more malignant form of inflammation. Again, in both cases the mitral valve was the seat of disease, although in the man's case the aortic valves were likewise affected. You will see from the specimens shown that the amount of proliferation on the valves was extreme, and the granular masses so produced were, in each case, readily detached, forming a basis for the ready production of embolism. Thirdly, in each case the onset of the fatal illness was sudden, and began with indications of brain disturbance, headache, and vomiting. They agreed in presenting pain in the left chest, breathlessness, and dyspnoea on exertion. In both the pyrexia was considerable, the temperature reaching 101° and 102° , though frequently falling below normal. In the woman it was noticeable that the anæmia, emaciation, and debility increased rapidly.

Cases of ulcerative endocarditis have been frequently mistaken for other diseases. Embolisms produce symptoms which vary according to the site. Sometimes a very fine embolism, if it be such as will pass the cerebral basic arteries, may produce disturbance of the cortex, resulting in convulsions or mania, as I showed by cases narrated at a previous clinical lecture, as well as hemiplegia; mania may result from ulcerative endocarditis. Again, these emboli sometimes get into the intestinal vessels, which they thereby plug, and this plugging may be followed by intestinal ulceration and hæmorrhage. This will go far to account for the fact that in some of such cases the suspicion that the disease is typhoid fever has been entertained, such diagnosis being all the more likely owing to the remittent character of the temperature. In some cases of ulcerative endocarditis there are changes in the character of the bruits, though that feature was not marked in the cases I have narrated. As certain granulations get washed off the valves the character of the sounds may be markedly altered.

In the case of the woman my diagnosis was based mainly on the following facts: The great debility, adynamia, loss of voice, the rapidly progressing anæmia; and, in the second place, upon the emboli, which evidently occurred separately,

first, in the brain ; then, in the spleen ; next, in the brachial artery, and probably in the kidneys.

In the man the opinion was led up to by the embol occurring in the foot, and by the continuous pyrexia without any lung or other inflammation sufficient to account for it.

The reason why, in these two patients, the old endocarditis took up this malignant form is not apparent. When the diagnosis of ulcerative endocarditis is made, the prognosis is extremely unfavourable, and probably all such cases end fatally.

CLINICAL LECTURE

ON

DYSENTERY.

Delivered at Westminster Hospital, January 15th, 1896,

BY

Surgeon-Captain J. H. TULL WALSH, I.M.S.,
Bengal.

As a clinical definition of Dysentery, we may say that it is a disease of the intestines, accompanied by pain, frequent desire to go to stool, tormina and tenesmus, with the passage of scanty motions, containing blood and mucus in varying quantities. Constitutional symptoms of a "febrile" nature are nearly always present, but vary greatly in severity. Dysentery is not contagious, but is no doubt infectious, and the excreta of a patient may contaminate drinking-water, and cause the disease in others. Dysentery "stools" should not be allowed to pass into the sewers, but should be mixed with earth and ashes, and burnt. Dysentery may occur in a "sporadic" or epidemic form. It is truly endemic in some parts of the world, and is more common in tropical than in temperate climates. This is not altogether a question of climate, but is influenced greatly by the fact that temperate climates are, generally speaking, inhabited by more civilised and more sanitary races. Although in the main banished from England by years of improvement in sanitary conditions, dysentery is not extinct, and "sporadic" cases, or even epidemics, still occur in country districts. Only last autumn I saw a severe case of catarrhal dysentery in a Norfolk village. I was asked to see a boy

who was reported to be suffering from severe diarrhoea. The case was made quite clear when I insisted that the "stools" should be passed into a chamber-pot, and saved for inspection. May I here be allowed to digress in order to impress upon you the necessity for personal examination of the material passed from the bowel in all cases of intestinal disease? Dysentery is most frequent in malarial localities, and this is the outcome of conditions favourable to both diseases—warmth, moisture, and organic and vegetable matter in a decaying state. I shall not have time to enter fully into the reasons for the "faith which is in me," but I am of opinion that dysentery arises more from the decay of animal than of vegetable matter, and that the *materies morbi* is of the nature of a *micrococcus*. As in the case of most diseases, so with regard to dysentery, it may be said that a person is more likely to be attacked under certain predisposing conditions. These are: rapid alterations and variations of temperature, dyspeptic conditions, however produced, diarrhoea produced by the ingestion of foul water, bad food, unripe fruit, etc. As regards age and sex, there is nothing to be said—all are attacked. Dysentery is very likely to occur in persons suffering from scurvy. Dwellers in tropical climates should always keep the abdomen warm.

Turning to the pathological aspect of dysentery, we find that it begins as a local disease in the mucous coat of the rectum and large intestine, an inflammation or colitis, affecting the solitary and mucous glands. In mild cases there is tumefaction, inflammation, and pain with excess of secreted mucus, often streaked with blood. A stage further we get extreme local congestion of the solitary glands, with ulceration and destruction of the intestinal mucous membrane, which is accompanied by more or less hæmorrhage. Further points in its pathology, and the constitutional symptoms, will be considered as we pass on to the question of classification. I shall consider three main forms of dysentery, which will include all others.

1. *Acute Catarrhal Dysentery.* Commencing with dyspepsia and pain in the abdomen. The pain varies in intensity, and is generally intermittent and of a "colicky" nature. Some diarrhoea is present, sometimes showing no signs of true dysenteric conditions for the first 24 hours,

during which time the condition is of the nature of a colitis, without effusion or ulceration. Next comes a frequent desire to pass a motion, with tenesmus and a burning sensation at the anus. The actual amount passed is usually small, and often contains no faecal matter, consisting only of blood-stained mucus. When the stools are more solid, as they may be, and scybala are present, the faecal matter will be coated with mucus and blood. Febrile symptoms are generally present, hot dry skin, furred tongue, thirst, and high temperature. Where there is much pain, the skin will often be moist and clammy, and not dry. In adults the temperature does not, as a rule, rise very high; but with children—in whom, by the way, high temperature often results from slight causes—it may range from 102° – 103° Fah. Vomiting is mentioned as a symptom by some writers; it is certainly not a common symptom in cases that have received no treatment. Left to itself, such a case might recover or might not, death in the latter case occurring from exhaustion, peritonitis, perforation, and shock, or from secondary abscess of the liver. Acute catarrhal dysentery may result from chill, from intestinal irritation, or, as in other forms, from a specific septic inflammation. It is often seen in children during the process of teething. It must not be forgotten that the presence of worms may produce in children, and sometimes also in adults, a condition resembling catarrhal dysentery. Intestinal parasites should, therefore, be carefully sought for. The lesions in this form are generally confined to the rectum and part only of the colon.

2. *Tropical Dysentery—Amæbic Dysentery—Septic Dysentery.* This, from a pathological and symptomatic point of view is only a more severe type of that mentioned. Such cases are more common in the tropics, and are accompanied by severe ulceration of the rectum and large intestine; and very rarely the disease may extend into the ileum. The ulcers may be small, circular, punched out, follicular, and very numerous, so that *post mortem* the bowel looks as if pitted with small-pox; they may be large, ragged, irregular and less numerous, with undermined edges. The mucous membrane at the seat of ulceration is partly or entirely destroyed according to the severity of the case, while the whole tube is infiltrated with inflammatory products and thickened, so much so

as to be frequently perceptible on palpation of the abdomen. Mucus and large quantities of blood are passed in severe cases, and hæmorrhage may at times be so severe as to result in death either directly, or indirectly by reducing the patient's strength. The mucus often comes away in masses resembling "casts" of the bowel, and actual sloughs are not uncommon. Under proper care such cases generally recover in a few weeks, the typical dysenteric conditions disappearing perhaps at the end of a week. It is, however, such cases that, when seen late, or indifferently cared for, produce a life-long misery in the form of chronic dysentery, or go to swell the roll of mortality. The causes of death are such as were mentioned under "Catarrhal Dysentery."

The name *Amæbic Dysentery* is most favoured by American writers, and was first suggested by Lambl in 1859. That these *amæbæ* occur in tropical dysentery cannot be denied. On the other hand I have seen the same *amæbæ* in cases of diarrhoea and in so-called healthy stools. *Amæbæ* occur freely in the stools of cows, pigs, and rabbits, and these animals do not suffer from dysentery. *Amæbæ* will no doubt more readily feed and multiply on a diseased surface, but biologically speaking they do not belong to the producers of putrefaction and necrosis, and it cannot at present be definitely stated that they alone cause this particular kind of dysentery. I am more inclined to agree with M. Zancarol, who, as the result of experiments carried out in Egypt, believes the cause of dysentery to be a *micrococcus*, probably a *staphylococcus*. The method of entrance into the body of such germs is generally through the use of impure water.

3. *Diphtheritic Dysentery.* Not in any way connected with diphtheria, but so called on account of the presence of false membranes and sloughs. The "febrile" symptoms are very marked, and there is much asthenia and frequently delirium. It is probably of bacterial origin, and occurs either as a primary disease generally in an epidemic form, or as a secondary disease. Dr. Bristowe was of opinion that it was frequently associated with pneumonia. This form is very fatal.

4. *Chronic Dysentery* is nearly always the result of a neglected subacute or acute attack of the "catarrhal" or "tropical" form. There is a continual uneasiness in the rectum and colon, irregu-

larity of the bowels with a diarrhoea which is generally only the diarrhoea of constipation, and is relieved by a mild purge. The bowel is much thickened, and a sluggish form of ulcer persists. The stools contain mucus in large quantities, and occasionally shreddy sloughs. Generally speaking, however, blood and sloughs are not common, but occur during exacerbations of the disease, should the patient relax any of that watchfulness over diet, rest and warmth, which is so absolutely necessary if we are to hope for recovery. Marked symptoms of dyspepsia are generally present with a furred tongue and unwholesome breath. Anæmia and emaciation are often very marked. The dyspepsia and weakness will continue even after apparent recovery, the glandular portion of the intestine is never renewed, and except as a channel for the passage of food, the large intestine becomes of little use.

Diagnosis.—I have not time to say much concerning the diagnosis in dysentery. If you have a firm grasp of the symptoms it should be fairly easy. Mild subacute cases and chronic cases seen by you for the first time might be mistaken for local affections of the rectum, such as syphilitic or malignant ulceration. An examination, together with the history and general constitutional symptoms will soon clear up your doubts.

Before commencing to treat any case of dysentery with drugs, put your patient to bed, and insist on a milk diet.

From the time that ipecacuanha was introduced into European practice from S. America it has generally been considered as more or less a "specific" in the treatment of dysentery, especially in acute cases. With the introduction of ipecacuanha, and no doubt dependent on it, the treatment by bleeding and mercury salts disappeared—the former for ever, the latter to be revived and maintained as useful in dysenteric conditions on account of the antiseptic and chologogue properties possessed by several of the mercury salts.

Ipecacuanha is an excellent remedy in acute cases when well borne by the patient. It should be given in large doses—20 to 40 grains. To counteract the nausea and vomiting which the drug may produce, 15 drops of tincture of opium, or a mixture of chloral hydrate and the bromide of potassium may be given some 10 or 15 minutes before the ipecacuanha is swallowed. In favour-

able cases ipecacuanha produces copious, frothy stools, which are followed by great relief to all the symptoms. Some years ago *ipecacuanha sine emetina* was introduced in the hope of obtaining a drug which would cure dysentery without producing nausea or vomiting. My own results with this preparation were not at all favourable, and, with many others, I believe that the emetin is the active principle which is useful in the cure of dysentery. Acting on this belief, I used, during the years 1890 and 1891,* a compound of emetin, with Mayers' reagent (HgI_2). This compound remains combined in the presence of acids, and so does not produce vomiting when introduced into the stomach. Further, we have the valuable antiseptic action of HgI_2 when separation takes place in the alkaline fluids in the intestines. In all, 39 cases were treated; one died, one passed out of hospital before cure could be accomplished. The rest recovered, and the average duration of dysenteric stools was about 4.5 days. In most cases, nearly a grain of emetin was taken daily—in powder with white sugar. In most of the cases the emetin powder was preceded by a castor-oil purge. The "stools" were similar in appearance to those passed under the ordinary treatment by ipecacuanha.

I now wish to draw your attention to some cases treated at the same time, and as control experiments, with "*Koorchee*," the bark, and "*Indra gau*," the seeds, of *Holanhena antedysenterica* (Wall). Ten cases were treated, and all recovered, with an average of 5.7 days for dysenteric stools. These figures do not mean that the patient was well and fit to get up or leave the hospital, but merely that the signs of acute dysentery had disappeared. *Emetin*, therefore, gives only slightly better results than *Holanhena*, and the difference might undergo alteration in another series of cases. We will now briefly consider some other drugs which have been reported as useful in the treatment of dysentery. In the *Indian Medical Gazette* for 1882, Dr. Owen reported 151 cases treated with milk and rice diet and tincture of aconite; 4.05 was the average time during which dysenteric stools lasted—all but one recovered. On referring to the Medical Annuals for the past three or four years, you will find some 20 or more drugs recommended for dysentery.

* *Indian Medical Gazette*, September, 1892.

Dolgopoff, of Kinsk, treated 140 cases successfully with *naphthalin*, a good antiseptic. Dr. Rennie (*Indian Medical Gazette*, 1886) advocated tinct. *cannabis indica*. Acting on a suggestion made in Bartholows' *Materia Medica*, Surgeon-Major Leahy (*Lancet*, 1890) treated 95 cases with a saturated solution of sulphate of magnesia and dilute sulphuric acid. This was given in small doses at frequent intervals, with the best results. This treatment has no depressing action on the system, and produces no nausea; it quiets and soothes the patient by relieving the hyperæmia of the gut, and probably frequently prevents ulceration. I have used this treatment for adults for three years, and consider it superior in every way to treatment by *ipecacuanha*. For young children and delicate adults I prefer a castor-oil emulsion, made as follows:—

Ol. Ricini	3j
Guin. Acac.	3iij
Sugar	3iij

3j–3iij every hour until the stools lose their dysenteric character.

The conclusion which I have come to is that we need not pin our faith to any particular drug in the treatment of dysentery, but that we must work on scientific principles, with such remedies as are suitable. Recalling to your minds the pathology as I have briefly sketched it, you will see that our aim should be to procure local cleanliness and rest for the diseased portions of the bowel. Cleanliness can be obtained by mild purgatives, the use of which should be discontinued after three or four days. When the stools are no longer “dysenteric,” give small doses of some antiseptic, and continue the fluid diet. Rest must be obtained by insisting on a fluid diet, and by keeping the patient in bed. Pain when great may require small injections of morphia, but generally passes off when the bowel is cleared of fæces, and at rest. Hæmorrhage is not usually severe, but it may in some cases cause the greatest anxiety. For internal remedies ergot and hazeline are the best. Fortunately we are often able to check hæmorrhage by injections into the bowel. Nitrate of silver (3j to Oj) forms a most useful injection, and several pints may be used. Some medical men treat most of their cases of dysentery with injections, even when there is no hæmorrhage. Quinine has been used freely

in injections of 1-1000 to 1-5000 in America, perhaps with a view to destroy *amæba*. The fluid diet should be continued for at least a week after the disappearance of blood and mucus from the stools. This careful dieting is most important; if properly followed out we should not find so many sad cases of chronic dysentery.

The treatment of chronic dysentery is almost entirely dietetic. Weak antiseptics may be given, and of these the liq. hydrarg. perchlor. in ʒss dose twice or thrice daily will be found most useful. Astringents should be avoided. There is another method of treatment which I will mention. I have never seen it adopted, and do not think that there are or ought to be many cases requiring it. I refer to colotomy, practised to give rest to the bowel, and to permit the direct entrance of injections into the large intestine. Reference is made to this form of treatment in the *Lancet*, 1895, p. 1578.

Of the sequels to an attack of dysentery stricture of the bowel and abscess of the liver are the most important. The former need not detain us now, but I must say a few words concerning the latter condition. I believe that the commonest cause of abscess of the liver is dysenteric or some similar and septic ulceration of the large gut. These abscesses result from a passage of septic matter along the portal vein. *During 1893 I collected some figures from the *post mortem* records of three large hospitals in Calcutta—the Presidency General Hospital, the Military Hospital, and the Medical College Hospital. Of 55 deaths 23 were from single abscess, 32 from multiple abscesses in the liver. Of the 23 single abscesses 15 were directly connected with dysenteric ulceration of the bowel, while of the 32 cases of multiple abscess 22 were connected with dysenteric ulceration.

* *Indian Medical Gazette*, July, 1893. I think the figures are correct, but quote from memory.

Dyspnoea of Phthisis.—Bernheim recommends the following:—

R. Caffeine citrat.	gr. iij
Sulphuric æther	ʒv.

Injected hypodermically Mxxx morning and evening.

AN abnormal condition of the abdominal viscera is the precursor of so many ills, that it is difficult to attach too much importance to the preservation of these organs in healthy action; and, in this connection it may be safely asserted that the use of drastic purgatives to remove ordinary intestinal obstruction has been very largely supplanted by mild laxatives, which, besides being, in many cases, equally certain in achieving the desired result, are much less distressing and inconvenient. Among such laxatives, natural mineral waters are fast superceding the use of drugs for this purpose. Perhaps the favourite of these is the famed product of the *Æsculap* spring in Budapest, and one need not wonder at this after perusing the following duly authenticated analysis:—

SALTS IN 10,000 PARTS OF WATER.

Sulphate of Potassium	...	0'104
Sulphate of Ammonia	...	0'061
Sulphate of Sodium	...	139'063
Sulphate of Magnesium	...	172'805
Sulphate of Calcium	...	20'788
Chloride of Sodium	...	29'047
Carbonate of Sodium	...	9'989
Carbonate of Iron	...	0'097
Carbonate of Manganese	...	0'429
Alumina	...	0'349
Silicic Acid	...	0'092

Total ... 372'824

Æsculap is not only markedly efficacious in dyspepsia, biliousness, constipation, and in toning up a sluggish liver, but its administration in fevers, nephritis, rheumatism, and neuralgia is attended by direct benefit. The water is remarkably uniform in composition, and this virtue, coupled with the fact that the bottling is carried out under English supervisors, who observe scrupulous cleanliness, goes far to account for its high standing with the profession, and will doubtless ensure progressive popularity.

THE connection between soap and medicine may at first seem remote, but in Dermatology and Midwifery the choice of an efficient soap, which cleanses without irritating, is rightly deemed of great importance, for the effect of soothing applications to delicate or inflamed skin would be neutralized by the employment of soaps containing

an excess of alkali. A point equally important is that the habitual use of super-fatted, pure soap will effectually ward off many skin troubles. But, for a reason well understood, medical men will not often be tempted into proclaiming what particular soap they find serviceable in practice, yet such knowledge is undoubtedly useful. It is certain that "*Vinolia*" soap is a great favourite with the profession—the unscented for ordinary use, and carbolic, coal-tar, sulphur, and terebene for skin affections. No praise could be higher, because, for any article to enjoy the esteem of the medical fraternity, it is almost superfluous to say that it must be pure and reliable.

THE foods which are prepared with the special object of affording—primarily to infants and invalids—the most suitable and concentrated nourishment requiring the least possible expenditure of digestive force, promise to almost rival, in numerical strength, the brands of tea. But, in the former case, if the practitioner is careful to obtain a copy of the certificates of analysis, there is no occasion for him to trust to the slow lesson of experience to enable him to exercise a wise choice. It is a trite maxim that "comparisons are odious" (some even assert that they are invidious), but it may be safely assumed in these matters that the mere fact of publishing comparative chemical analyses proclaims that the product possesses those virtues which most ensure success. This seems to be the case with "*Nutroa*." Analysis:—

	"Nutroa" Food completely dried.	The "Ideal Diet" of Professor Halliburton.
Proteids	19'64	20'25
Fat	12'12	12'15
Carbo-hydrates.	67'02	67'50
Phosphoric Acid	0'69	
Other mineral matter	0'53	
	<u>100'00</u>	<u>99'90</u>

The reader will see how closely "*Nutroa*" approaches to the recognized standard of Professor Halliburton. Perhaps the chief point about the food is the large proportion of the important constituent fat, which is so essential for the repair or

building up of tissue, both in infant and adult. The ease with which this preparation is assimilated has been clearly demonstrated by actual experiment, and the available evidence of its effects upon those for whom it is specially prepared gives emphatic support to the flattering report of Mr. Wm. Jago, the well-known analytical chemist. This gentleman declares that "Nutroa" "stands in the very highest rank of all food preparations I have ever examined."

It may be truly said of the remedies available for the cure or relief of disorders of the digestive tract that "their name is legion, for they are many," but who will assert that they are too many? Yet, the most gratifying fact is that, generally speaking,

each new product marks a distinct advance towards perfection. Diastol, the recent emanation of "The Standard Malt Extract Co." indeed "comes as a boon and a blessing to men" with digestive troubles. It is evolved with great care from the finest malted barley, the mineral salts being preserved, while an excess of soluble carbohydrates is avoided. It will thus be evident that it is calculated to suit all persons who have a tendency to produce an uncomfortable amount of adipose tissue, and we can recommend all sufferers from indigestion to try its effect in conjunction with a reasonably regulated diet. As this preparation is many times richer in diastase than the average malt extracts, a teaspoonful twice a day will be found very efficacious.



The Association for the Supply of Pure Vaccine Lymph,
12, PALL MALL EAST, LONDON, S.W.

SOLE AGENTS FOR

DR. WARLOMONT'S CALF VACCINE.

Tubes, 2s. each. Half tubes, 1s. each. Pomade in Vials, 5s., 10s., 20s.

HUMAN VACCINE (from healthy children only, microscopically examined and source quoted). Tubes two-thirds full, 1s. 8d. each. Tubes one-third full, 1s. each. Tubes two-thirds full (same as those mentioned above, but without source), in quantities for export, 25 per 100 Tubes. Pin Points (uncharged) gratis with Vaccine if desired, or 1s. per dozen. Vaccine Ejectors, 1s. 3d. each, including postage.

Office hours, 10 to 4; Saturdays, 10 to 2. P.O.O.'s (including postage, and crossed "London and Westminster Bank") with orders, payable to
EDWARD DARKE, Secretary.

Special Analytical and Biological Commission

ON THE

MILK SUPPLY OF LONDON.



The *British Medical Journal* of July 27, 1895, says:—

"According to the report of our Commission, the 'MILKMAID' brand contains 990 PER CENT. MORE BUTTER-FAT than is contained, on an average, in the other brands examined. This is a fact of great importance to the public, and should also serve as a guide to the medical profession when prescribing condensed milk. We have taken the 'MILKMAID' brand as a standard because it is the original condensed milk introduced into Europe about thirty years since, and its uniformly high reputation has helped to maintain the demand for such a product."

SAMPLES FREE TO THE PROFESSION.

ANGLO-SWISS CONDENSED MILK COMPANY,
10, MARK LANE, LONDON, E.C.

THE CLINICAL JOURNAL.

WEDNESDAY, FEBRUARY 26, 1896.

CLINICAL LECTURE

ON

MALIGNANT DISEASES OF THE LARYNX.

Delivered at St. Thomas's Hospital, Jan. 14 and 31, 1896,

BY

FELIX SEMON, M.D. Berl., F.R.C.P. Lond.,

Physician for Diseases of the Throat to the Hospital.

GENTLEMEN,—The subject of to-day's lecture is malignant disease of the larynx. Although this terrible disease is, fortunately, rare, I wish to interest you all very particularly in it. My reasons for this are as follow:—First, that our views with regard to the diagnosis and the treatment of malignant disease of the larynx have, during the last ten years, undergone very considerable changes for the better; in so far that, in a large number of cases we are now able to diagnose the disease much sooner than we were previously able to do. Secondly, because the *technique* of the operation has been improved, resulting in a much larger proportion of the patients suffering from malignant disease of the larynx being spared fatal termination of the disease; and thirdly, because in order to obtain all those benefits which accrue from improved diagnosis and improved methods of operation, one thing is absolutely essential, namely, that the general practitioner in whose hands these patients naturally are during those very stages of the disease in which radical operation promises success, should be able to diagnose the disease himself, or at any rate should not neglect those early stages, and should avail himself of the experience of those who, by special training, are enabled to form a diagnosis at the earliest possible time. As I have stated on another occasion, nothing is sadder than to see these patients at such a late stage of the disease that only palliative measures can be suggested, with the feeling constantly before one's mind that if they

had been seen earlier they might probably have been saved. The possibility of rendering them that invaluable service is in the hands of the general practitioner, and therefore I invite your very earnest consideration to all I am going to say with regard to malignant disease of the larynx.

First, let me refer to the etiology of the disease. I need hardly say that we are in the same uncertainty here as we are regarding the origin of malignant disease anywhere in the body. Whether it will ultimately be found to be a microbic affection of some kind or other, or whether it is merely, as it were, a developmental error which leads to the formation of malignant growths, is at present still an absolutely open question. A few facts, however, are known, and these are of great importance to the question. In the first place, you will find that practically in all cases of malignant disease of the larynx the disease is *primary*, that is to say, it originates within the larynx itself, or at any rate in its immediate neighbourhood, and attacks the larynx by *contiguity* only. Indeed, so far as I know, there is practically not a single case of positively ascertained *secondary* or *metastatic* cancer of the larynx on record. The reason of this is, in my opinion, to be found in the peculiar anatomical arrangement of the lymphatics of the larynx, to which I drew your attention in the first lecture of this term, when speaking of the anatomy of the larynx generally. According to the researches of Professor von Luschka, the lymphatic supply of the larynx in so far differs from that of its neighbourhood that, although the larynx is richly supplied with lymphatic vessels, they form, as it were, a net-work of their own, and do not freely anastomose with the lymphatics of their neighbourhood. In that respect they are entirely different from the lymphatics of the pharynx, which, as you know, freely anastomose with those of the neck, and indeed with all the neighbouring lymphatic vessels. Again, the laryngeal lymphatics empty themselves into four small lymphatic glands, two of which are situated at the sides of the trachea, and two just below the superior horn of the hyoid bone. This lymphatic

arrangement at once explains the otherwise very unintelligible difference in the participation of the lymphatics which is met with in certain diseases which may attack either the pharynx or larynx primarily. One of these diseases is diphtheria. You know that in pharyngeal diphtheria one of the first signs usually is a very considerable engorgement of the cervical lymphatics, and the explanation is the one I have just given you—the lymphatics of the pharynx anastomose with the lymphatic vessels of the neck, and in consequence of this the infectious material is, at a very early period, carried into those parts, and leads to secondary enlargement of the cervical glands. In primary laryngeal diphtheria, on the other hand, we find that although the whole larynx may be lined with false mucous membrane, there is, in many cases, no enlargement of the cervical lymphatics at all. Exactly the same obtains of pharyngeal and laryngeal cancer. Malignant disease of the upper part of the throat, whether starting from the pharynx proper, or from one of the tonsils, usually is characterised, at a very early stage of the disease, by very considerable tumefaction of the cervical lymphatics, corresponding to the seat of the internal malignant disease. In laryngeal cancer, on the other hand, the lymphatics may—and often do—remain free until the very end of the disease; that is to say, until the patient's death. The importance of this fact is very great indeed, not merely with regard to diagnosis, but, more important still, with regard to treatment; because, so long as the malignant disease remains limited to the larynx proper, you have a reasonable chance, unless it be too far advanced, to cure by extirpation the originally local disease. From the moment, however, that the cervical lymphatics have become secondarily affected, the chances, even if good with regard to immediate survival from the operation itself, are infinitely worse with regard to the all-important question of recurrence. You therefore see that the arrangement of the lymphatics is a point of paramount importance. I am perfectly aware that this lymphatic supply, as first described by Luschka, has been the subject of controversies, and that Sappey and his followers have contested it. But I feel justified in giving you this description, because in every respect it agrees with the clinical facts, and, above all, with the very point from which I started—that real

secondary or metastatic cancer of the larynx is practically unknown.

We classify malignant disease of the larynx according to whether it originates within the larynx proper, or attacks that part by contiguity, and the most generally accepted division of the subject is the one proposed by the late Professor Krishaber of Paris. It is of practical value because, in the present state of our knowledge, it is a means of deciding which cases are best suitable for radical operation, and which are less so or not at all.

To the first class belong cases of what Krishaber called "intrinsic" laryngeal cancer; that is to say, cases in which the original seat of the disease is within the larynx proper; to the second, cases of "extrinsic" laryngeal cancer, in which either the vestibule of the larynx, the epiglottis, the aryteno-epiglottidean fold, or the posterior surface of the cricoid cartilage is affected; or in which the disease, starting from the pharynx or neck, invades in its progress the larynx anywhere. Considering the anatomical facts, this division, as you see, is not an academical one, but of immediate and great practical interest.

With regard to the forms in which laryngeal malignant disease appears, we meet the two large classes of malignant disease which are found anywhere, namely, carcinoma and sarcoma. Of these, the carcinomatous form is infinitely the more frequent, sarcomatous disease of the larynx being decidedly very rare. As to the forms of carcinomata which we meet with, it can be confidently stated that the epitheliomatous or squamous-celled variety is that which occurs by far most often; the medullary form, scirrhous, or columnar-celled carcinoma, being in an absolutely insignificant minority.

With regard to the question of sex, it is very remarkable that whilst the difference with reference to sex in benign neoplasms of the larynx is, comparatively speaking, a small one, in malignant disease of the larynx the male sex is, according to all good statistics, infinitely more liable than the female. But the causes of this difference have not been ascertained. It is usually thought that, amongst other causes, alcohol and nicotine may be productive of malignant disease of the larynx in that chronic laryngitis and pharyngitis induced by the over-use of stimulants in those otherwise predisposed, may later on form a favourable element in the ultimate production of laryngeal malignant

disease. I am not prepared to deny this *in toto*, but speaking from large personal experience, I can at any rate give you the somewhat reassuring statement that, so far as smoking is concerned, I have not been able to make out any particular predisposition of those who smoke as against those who do not. I mention this more particularly because for some time it has been a fashion amongst some, to threaten those who are addicted to the weed with the possibility of the development of "cancer of the throat." That, naturally, in a large number of cases—particularly in private practice, of which I have full notes—there were a few excessive smokers, goes without saying, and so does the fact that there were many more who smoked moderately, but there were also a comparatively large number of people who had never smoked at all, or so little, that no unbiassed person could for a moment consider this as an etiological factor in the production of the disease. Altogether, these influences have been, I think, considerably over-rated. The same, I may say, obtains with regard to professional voice-use, which has been set down as a cause of malignant disease of the larynx. In a paper which I published in the *Lancet* last year, I investigated the occupations of the various persons who had been under my care afflicted with laryngeal cancer. I could not say that I found any preponderance on the part of these avocations in which the voice is frequently used over those who did not use their voices professionally at all. Therefore, the cause of the great preponderance of males in cases of cancer of the larynx is still clouded with considerable obscurity.

Then with regard to age. As you know, age is a very important element in the occurrence of cancer altogether, and it is doubly important in the present question as relating to the differential diagnosis of malignant from benign growths, because—as you will hear later—cancer of the larynx in its first stages not infrequently appears entirely in the form and aspect of an innocent laryngeal neoplasm.

The general experience with respect to cancer, namely, that it is an affection of middle or advanced life also entirely obtains in the larynx. There can be no doubt that the enormous majority of all cases of laryngeal cancer are met with between 40 and 70 years of age, or, more pre-

cisely speaking, between 50 and 70; but neither young nor very advanced ages give immunity. I have seen one patient of 26 whose laryngeal growth was microscopically proved to be malignant, and another patient, aged 35—but almost all others have been above 40. On the other hand, I have myself seen several cases in which the patients were over 80, and I have described the case of the oldest patient in which malignant disease of the larynx has ever been observed—a man aged 83.

We now come to the question of symptoms. I will first discuss the subjective, and afterwards the objective symptoms of the disease. With regard to the former, I wish you to disabuse your minds at once of certain statements which you find, even at the present time, in the majority of text-books concerning the subjective symptoms of malignant disease of the larynx. There you usually read an imposing array of symptoms which, if they were all present in a case under observation, would indeed make the diagnosis an easy one. You are taught that laryngeal cancer is characterised by hoarseness, aphonia, difficulty in breathing and swallowing, by considerable pain in the throat—often extending into the ears—by *stœtor* of the breath, by repeated hæmorrhages, by swelling of the cervical glands, and by general cachexia, which latter is one of the characteristics of cancer generally. Now I certainly do not deny that you may and do find all these symptoms in the *last* stages of the disease—that is, in those which we can no longer help; but what I wish you to take to heart is the memorable absence of almost all those symptoms in a very large number of cases during the early stages; in other words, whilst real help can be afforded.

My own experience has shown that the vocal cords in the cases of intrinsic malignant disease are by far the most frequently affected parts of the larynx in the early stages of the disease, and that *the* one symptom which is invariably present in these cases is hoarseness. I cannot sufficiently emphasize the fact that such hoarseness may exist many months—in some cases for a year or more—*without a single other symptom supervening!* Therefore I believe it is the duty of every general practitioner in a case of persistent hoarseness in a person of middle age, without any other symptom whatever being present, to make a laryngoscopic examination himself; or if he cannot, to have it

made by someone who can, in order to ascertain whether this hoarseness is due to a harmless affection, or whether it is the first sign of malignant disease. This, surely, is not asking too much.

Supposing the tumefaction which forms the original symptom of the disease to start from a vocal cord, the disease at first usually runs a very slow course in the great majority of cases, and many months may and often will pass before the hoarseness passes into more or less complete aphonia. This stage will of course be reached as soon as the tumefaction has attained such a size that it interferes not only with the vibrations of the vocal cord to which it is attached, but also mechanically hinders the vibration of the opposite vocal cord. In such cases there will be, besides the vocal disturbance, as a rule, also, from the narrowing of the chink of the glottis, more or less considerable dyspnoea, which ultimately may necessitate tracheotomy.

We now come to the question of dysphagia. Unless the disease primarily starts, say, from the epiglottis, from the aryteno-epiglottidean folds, from the posterior surface of the cricoid cartilage, etc., or unless it makes its way later on into the food-passages, there need not be—and in many cases there is not—difficulty in swallowing. Everything in this respect depends on the original seat of the disease, or upon what parts associated with the performance of deglutition are invaded later; but it is by no means characteristic of laryngeal cancer, *per se*, that there should be any dysphagia whatsoever.

Exactly the same obtains—and this is a very important point—with regard to the question of pain. It is, even nowadays, a sort of axiom that laryngeal cancer is one of the most painful diseases existing. This is by no means a true generalisation. Of course I do not deny that there are cases—and, indeed, a good many—in which pain is a prominent symptom, or in which pain may be altogether the first symptom; but this again does not depend on the disease *per se*, but upon the question whether one or another of the sensory nerves of the part are, at an early period, implicated in the disease. As soon as the internal branch of the superior laryngeal nerve becomes involved, you will of course find pain, just as in any other disease where that nerve is implicated. But this pain need never occur, and

I have seen many patients suffering from laryngeal cancer, in whom up to the time of death there was no pain. Professor von Ziemssen at one time thought that early pain in the throat, when shooting to the ears, was one of the characteristic symptoms, and, indeed, almost pathognomonic of malignant disease of the larynx. This extension of throat-pain to the ears is a reflex-phenomenon. The superior laryngeal nerve being irritated, the irritation is transferred to the auricular branch of the pneumogastric nerve; hence pain is experienced in the ear. This pain, however, is not, as it was at one time believed to be, pathognomonic, but may occur in any affection implicating the superior laryngeal nerve, and as a warning not to trust to it too much, I may relate at this juncture a case in my own experience.

In 1879, when we were all under the influence of von Ziemssen's teaching, I saw, at the Throat Hospital to which I was then attached, a married woman, aged about 60. She complained of shooting pains from the throat into one ear; there was a considerable ulcerating tumefaction on the corresponding side of the larynx, and she looked very cachectic. Having regard to her age, to the cachexia, to the absence of any syphilitic history, and more particularly to the pain shooting into the ear, the diagnosis made was that of malignant disease of the larynx, and tonics were prescribed. I never saw that patient again alive, but two or three months afterwards I was present at the autopsy, and, to my deepest regret, it was found that the patient was not suffering from malignant disease, but from tertiary syphilis of the larynx and trachea, there being at the same time some big gummata in the liver. That case made a never-to-be-forgotten impression upon me, the more so because it is usually taught—quite rightly—that syphilitic disease of the larynx is an almost painless affection, and yet in this particular case the shooting pain to the ears was the most prominent symptom! I wish to spare you a similar unsatisfactory experience, and I hope you will remember the case. However certain you may be that the disease is malignant, do what you should in every ordinary case in which there is the least uncertainty—give iodide of potassium in large doses for a fortnight, before definitely pronouncing on the nature of the disease.

Another subjective diagnostic sign is *foetor*.

Fœtor need not be present for a long time, so long as there is no breakdown of the malignant tumefaction. It is only when actual ulceration occurs that fœtor of the breath is present, and that rather rarely occurs in an early stage.

Exactly the same may be said concerning hæmorrhages. When hæmorrhages occur, particularly slight and often repeated ones, no doubt they are very characteristic of malignant disease of the larynx; but a good many cases ending in death have been reported, in which no hæmorrhage during lifetime had ever been encountered.

I have already spoken on the question of engorgement of the lymphatics.

As to cancerous cachexia, I may tell you that sometimes patients look as strong and healthy as they possibly could until the very end of their lives, without any sign of cachexia being manifested. I remember the case of an old solicitor some years ago, who was in this hospital under the care of my late lamented colleague, Dr. Bristowe. This patient had long ago been tracheotomized, and the malignant affection completely filled out his larynx, and yet he looked as rubicund as if he had been breaking his several bottles of port every day, and had nothing whatever the matter with him. Yet that patient had been suffering for at least two years from malignant disease of the larynx.

I lay great stress upon all these points, Gentlemen, because I do not wish you to be led astray by mere doctrinary teaching, or to neglect the early stages of the disease because of the absence of any symptom but hoarseness.

I now come to the objective signs of malignant disease of the larynx. The disease appears in many different forms. First, in the initial stages it may appear in the form of simple congestion of the part in which afterwards tumefaction occurs. Already, when discussing tuberculosis and syphilis of the larynx, I reminded you that isolated congestion of one vocal cord usually is a sign of serious omen. That point is now quite universally admitted, and is of great significance with regard to laryngeal cancer as well. But even in cases in which the congestion is bilateral you must not at once rush to the conclusion that it can be nothing but a simple catarrhal laryngitis. Two years before his death, I saw a very well-known public man, who sought advice on account of

hoarseness, which was chronic, having lasted—according to his own statement—a good many years. Nothing abnormal was detected besides general congestion and thickening of both vocal cords, and I diagnosed chronic laryngitis. A few weeks afterwards he applied to a Life Assurance office to insure his life for a large sum. The insurance physician, who happens to be a very excellent laryngologist, was made suspicious by the patient's hoarseness, examined his larynx and found the same bilateral congestion which I had noticed a few weeks previously. He, too, believed it to be a case of chronic laryngitis, and knowing that this affection does not at all curtail life, he had no hesitation, as the patient was otherwise well, in accepting his "life." A few months afterwards unmistakable signs of malignant disease of the larynx appeared; the disease took a very rapid course, and the patient died, I believe, within a year of being insured. It was lucky that I had seen the patient previously and had notes of the case, so that I could come to my friend's aid and assure the directors that—from the symptoms then present—no one could have judged that there was anything present except chronic laryngitis. That case shows how careful one ought to be; and that even bilateral congestion of the larynx—though apparently catarrhal—does not exclude the possibility of the disease being malignant.

Whilst in a number of cases simple congestion is the first sign, in others the disease begins at once in a form of diffuse tumefaction. This may originate from the vocal cords, from the ventricular bands, from the epiglottis, or, indeed, from any other part of the larynx, but in the greater number of cases the vocal cords and the ventricular bands are the original seats of the disease. This tumefaction, which at first may show itself simply as a general thickening of one of the vocal cords, gradually extends, and later on the whole of one side of the larynx may become completely involved, with the result that instead of seeing the pre-formed parts, you are able to discern nothing but a diffuse tumefaction of the whole corresponding side of the larynx. I show you here some excellent specimens of this. Cases of this sort, if seen in an advanced stage of the disease, do not allow the observer to form any definite conclusion as to the starting-point of the disease.

Such cases, in which a large part or almost the

whole of the larynx is changed into one mass of infiltration are not difficult to diagnose; the only mistake possible would be in confusing them with gummatous infiltration, and my previous advice to give iodide of potassium in all cases therefore obtains in them with particular force.

In another series of cases the disease presents itself from the beginning as a globular, sessile, somewhat irregularly nodulated mass, the colour of which may be that of the surrounding mucous membrane, whilst in other cases it is either paler or more dusky than the latter. This class, particularly when the tumefaction has attained a certain size, usually does not offer special diagnostic difficulties.

The really difficult cases, however—and they are numerous enough—are those in which malignant disease appears at first in the form of an apparently innocent tumour. These are the cases about which, in modern times, so much controversy has been raging. You have all heard of the great discussion which was engendered at the time of the illness of the late Emperor of Germany, about the question of transformation of benign growths into malignant ones, and more particularly under the influence of intra-laryngeal operations. If it were true that by instrumental interference with the larynx one could change an innocent growth into a malignant one, it would mean—as I said at the time, and as I now fully maintain—the death-knell of intra-laryngeal surgery, for no honest operator would plunge his forceps into the larynx of any patient if there were just reason for the fear that he might thereby convert an innocent growth into a malignant one. The truth of the matter, however, as shown by the collective investigation which I made at the time, and the results of which are now generally accepted, is this:—In the majority of all cases of so-called transformation the disease was malignant from the very beginning, but was not recognized as such. As in other parts of the body so also in the larynx, malignant disease may show itself in the form of an innocent papilloma. (As an analogous case I may mention the bladder, in which the differential diagnosis between benign and malignant growths often is a matter of great difficulty.) Again, malignant disease of the larynx may occur in the form of a simple fibroma, and even, in some rare cases, the malignant growth may be completely pedunculated. I show you here a case from the museum, which

illustrates this remarkably well. The real growth is entirely surrounded by a shell of part fresh and part organized blood-clot, which latter, during life, quite obscured the true structure of the neoplasm, giving rise to the appearance of an angioma. Within four months after removal the growth recurred, and it was then noticed that though still pedunculated its base was somewhat elevated. Repeated microscopic examination now demonstrated the malignant nature of the disease. This variety is extremely rare, this being the only case of the kind recorded.

In this second specimen you see a broad-based, papillomatous-looking mass protrude from the ventricle of Morgagni. The first microscopic examination of a fragment intra-laryngeally removed seemed to demonstrate the innocence of the growth; the second, made within five days from the first, conclusively proved its epitheliomatous character. The affected half of the larynx was removed, the patient made a splendid recovery, and died 6½ years later from heart disease altogether unconnected with his previous laryngeal affection. From such cases you will at once see that however eminent a microscopist you may submit a particle to, in the nature of things this is usually taken from the periphery only, and may not include any of the real central disease. The verdict of the pathologist however, obviously, can only be based on the actual piece submitted to him. Non-observance of this simple fact has more than once led to very sad results. And further in such cases, because the microscopist has in the first instance pronounced the growth to be benign, and it has subsequently been proved to be malignant, you are not entitled to say, "This was primarily benign, but has lately become malignant, possibly due to instrumental interference." That would be a logical fallacy of the very worst character! I am not going to absolutely deny that there is a possibility of benign growth anywhere in the body undergoing, later on, a malignant change; we see it in all parts, why should it not occur in the larynx? The best example I can refer to is that of the hypertrophic thyroid gland. Here malignant changes occur much more frequently than in the normal thyroid gland. Now, just as goitre is a hyperplastic process of the formative elements of the gland, thus also, benign growths of the larynx represent a true hyperplasia of pre-existing tissues of the larynx, and

there is not the least reason why malignant degeneration should not take place in such hyperplastic formations here as well. It would be ridiculous to altogether deny that possibility. Such cases, however, we find to occur *extremely rarely*, and the collective investigation to which I have repeatedly referred, affords not the least evidence that any kind of instrumental interference has played the slightest part in bringing about such a change, it being on the contrary, most remarkable that such malignant changes have been reported more frequently in proportion in those cases of laryngeal growth in which *no* operative interference had been performed than in those which had been operated upon. We can therefore safely reject the bugbear of the malignant transformation of benign growths in the larynx from instrumental interference, and it will be much more in harmony with actual facts if we assume that in the first instance a diagnostic error has been made, which, even to the most experienced eye, it is sometimes extremely difficult or even impossible to avoid.

Whilst this must be freely admitted, there are, on the other hand, certain points which will assist us in establishing this differential diagnosis.

First of all, with regard to papilloma. As I told you when speaking of benign growths of the larynx, Virchow has taught us that benign growths, if occurring on the vocal cords, have a tendency to localize themselves on their *front* parts, and very frequently on the anterior commissure of the vocal cords. If, on the other hand, you find a papillomatous-looking growth on the *posterior* parts of a vocal cord, particularly if the patient be well advanced in years, that is primarily suspicious, and still more so if such growth be seated in the inter-arytenoid fold, where benign growths practically never occur. And further, when an apparently innocent papilloma springs from a part in which it is unusual to find benign growths, such as the epiglottis or the aryteno-epiglottidean fold, the case is at least suspicious. In the specimen I just showed you, there was no other evidence of malignity whatever, but the neoplasm originated from the aryteno-epiglottidean fold, where such growths practically never occur, and my suspicions were at once roused. When first reporting the case, together with Mr. Shattock, to the Pathological Society, we therefore observed the precaution of

describing it as an "anomalous tumour" of the larynx, and not as a papilloma, implying thereby that we left it an open question as to whether it was benign or malignant. The further course of the case, as already mentioned, fully justified my suspicions.

A second point of practical importance with regard to the differential diagnosis between genuine papilloma and malignant disease appearing in the form of papilloma is as follows:—In genuine papilloma the apices are more or less *rounded*, whereas, in malignant disease simulating papilloma, I have observed that the individual projections of the growth are very much *pointed*; and further that the colour of the suspicious growth occasionally is nearly snow-white, much whiter than ordinary papillomata, which usually show a slightly pinkish hue. I cannot give you a better comparison than that such a growth looks like a sort of snowy meadow. Another suspicious sign is that if you find—as I have seen—a papillomatous-looking growth occupying the whole edge of one vocal cord, whilst the other vocal cord is perfectly free, and particularly if this extends far backwards, where benign growths are very rarely, if ever, found.

Now, with regard to the mobility of the affected vocal cord. Benign growths of the larynx, however big they may be, do not intrinsically cause an impairment of the movements of the vocal cord on which they are planted. I need hardly say that if a large growth from its mere size almost fills up the glottis, it will mechanically prevent the juxtaposition of the cords on attempted phonation; but if the other vocal cord could be taken out of the way, the adduction and abduction movements of the affected vocal cord would be perfectly free. In malignant growths, on the other hand, we actually see, as a rule, the smaller part only of the disease, the greater part being in the mother tissue itself, which it infiltrates. This consideration enables us to understand that if malignant disease takes place on the vocal cord, even if the disease may apparently not be very extensive, we usually meet, even in early stages, with impaired mobility of that vocal cord. It does still move in respiration and phonation, but more slowly and languidly than the other vocal cord. I drew attention to this years ago, as one of the most important signs in the differential diagnosis

between benign and malignant growths, and although the statement received much opposition it has been corroborated by a number of the most competent observers. At the same time I fully admit that this symptom need not always be present. There are cases of malignant disease of the larynx which are comparable to rodent ulcer on the face; that is to say, the disease is of a more superficial character; and so long as it remains superficial no slowness or slackness of the movements of the vocal cord need take place. How careful, however, one has to be with regard to this point will appear from the following case:—

A patient came to me with the whole of his left vocal cord apparently embedded in a sort of white papillomatous fringe, and from the very first I entertained a grave suspicion of malignancy. The patient at that time was 56. I removed small pieces of the excrescences with Mackenzie's forceps and submitted them to Mr. Shattock, who examined them, and came to the conclusion that they were of papillomatous nature. My clinical fears, however, were not allayed by the microscopical examination, and I explained to the patient that I must make another exploratory excision. He agreed, and five days afterwards I removed another portion, this time going much deeper. Mr. Shattock, after examination, at once pronounced this to be epithelioma with papillomatous excrescences on the surface. Not even the most fanatical believer in the transformation of benign growths into malignant ones by instrumental interference could quote, I venture to say, this particular case to be an instance in support of his theory. The reason, however, why it is quoted in this connection, is a different one. I was already, at that time, very deeply imbued with the notion that early deficient mobility of the affected vocal cord was one of the most important signs of laryngeal malignant disease. In this case the whole vocal cord was apparently embedded in this tumour, and yet the arytenoid cartilage moved just as frankly as the other. I had, therefore, to face the dilemma that this case appeared flatly to contradict my own teaching. Nevertheless, after the second microscopical examination thyrotomy was performed, and a large tumour was found growing, not from the left vocal cord, as had seemed from laryngoscopic examination, but from the left

ventricle of Morgagni. 'The left vocal cord, which had been completely covered by the growth, was quite healthy, and its free mobility was thus satisfactorily accounted for.

Such being the initial objective signs and subjective symptoms in the initial stages of the disease, whether it presents itself in the diffuse or in the more circumscribed form, we have now to study the phenomena of its further progress.

Broadly speaking, the latter may be characterised as one of *continuous*, usually at first rather slow, later on more rapid advance. Of this rule, however, important exceptions occur. On the one hand, in some cases the disease, after reaching a certain stage, sometimes appears to become temporarily arrested, and this occasionally for many months, nay, even for one to two years; on the other, in a few cases an apparent, though unfortunately fallacious, considerable improvement seems to take place.

Such cases, even to the most experienced observer, sometimes come as a complete surprise, yet the apparent improvement is easily enough explicable. Supposing you find a growth, with more or less diffuse infiltration, starting from one vocal cord, say the left, the right vocal cord being perfectly healthy. When the two cords come together during the act of phonation the tumefaction of the left vocal cord will not merely interfere with the vibration of that cord, but with the vibration of its fellow also, by simply mechanically preventing its free vibrations, and hence more or less complete hoarseness or aphonia will ensue, and will become greater as the tumefaction of the part increases. Of course, a stage will be reached when the disease will practically infiltrate the whole left half of the larynx. Then there will be not merely complete aphonia, but, in consequence of the great encroachment on the glottic space, there will be also considerable dyspnoea. The diagnosis may have been definitely established by intra-laryngeal removal and microscopic examination of a fragment, and you know that you have to deal with a case of epithelioma.

After this you perhaps may not see your patient for two or three months, nor hear anything of him, and may think he has gone from bad to worse. One day, however, the patient may come into your room speaking in almost a normal

voice, as has twice happened to me, and with the dyspnœa gone. I assure you it is startling, especially if you have had histological corroboration as to the malignancy of the disease. The patient may even look much better than formerly and feel more hopeful. What has taken place? You know that in malignant disease of most parts the original tumefaction gradually breaks down, and the breaking down occurs in the periphery first; not like gummata, which generally soften and break down centrally first. This peripheral breaking down then will, at some period, result in what appears to be a simple ulcer, the outline of which conforms more or less to the former outline of the vocal cord. When that stage is reached, the vibrations of the other vocal cord will no longer be interfered with, and experience shows us, in partial extirpation of the larynx, that when one vocal cord is unhampered, the voice may be almost normal. At the same time, in consequence of the diminution of the encroachment upon the glottis, the air is allowed a freer entrance, so that the dyspnœa will disappear. I need not tell you that this apparent improvement is, unfortunately, very fallacious, and a very temporary one; the growth breaks down more and more, fresh tumefaction, as a rule, appears in its periphery; gradually, the previously unaffected side becomes invaded as well, and finally, the whole larynx may be one mass of ulceration and tumefaction.

As soon as the disease extends outside the real confines of the larynx, and the pharynx is involved, engorgement of the cervical lymphatics usually soon takes place, and an enormous mass may form at one or both sides of the larynx. If tracheotomy has been performed, but not sufficiently low down, you may find that the malignant disease makes its way along the anterior wall of the trachea into the wound, resulting in a luxuriant growth, with alternating tumefaction and breakdown round the tracheotomy tube. Finally, there may be such an appearance as I show you in this museum specimen, *i.e.*, the tube lying in a large gaping hole. I need not tell you that progress in this direction is not the only possible one. In a number of cases the disease advances to the posterior, *i.e.*, the œsophageal surface of the cricoid cartilage, and, under these circumstances, grave difficulty in swallowing is produced.

A few words are here required concerning that form of extrinsic laryngeal cancer, in which the course of events is exactly the opposite one to the form last described, *i.e.*, in which the disease *starts* from the œsophageal surface of the cricoid cartilage. It is, perhaps, the most cruel variety of all, being not only from its situation and the early infection of the neighbouring cervical lymphatic glands the least suitable for radical operation, but engendering sometimes, in addition to dysphagia (and often pain), early severe dyspnœa by mechanically destroying the muscular substance of the abductors of the vocal cords, the posterior crico-arytenoid muscles, which are situated just underneath its starting-point, and by thus causing bilateral paralysis of the glottis-openers and necessitating early tracheotomy.

Again, it is characteristic of malignant disease that no tissue is spared in its relentless onward march. Thus not only mucous membrane, muscles, nerves and vessels, but also the cartilages may become affected, and perichondritis, caries, and necrosis of the laryngeal framework may supervene, which in turn may so completely overshadow the symptoms of the original disease that—even to the most experienced eye—it is quite impossible to say to what the perichondritis is due. Instances of that kind are by no means rare, and I show you here an example in a specimen from the museum. That particular case had been originally described as primary idiopathic perichondritis by another observer. Tracheotomy had been performed, a swelling appeared in the neck round the tube, and this was actually incised in the belief that it represented an abscess in the neighbourhood of the tube. No pus, however, escaped after the incision. The patient then came under my observation, and at that time I must say it was impossible to decide from laryngoscopic examination what the enormous œdematous swelling seen inside the larynx was due to. But of course an external swelling immediately raised the suspicion of malignancy. The patient was taken into the hospital under Sir William MacCormac's and my own joint care. A small piece of the external swelling was removed, which on microscopical examination turned out to be unmistakable epithelioma. In this case perichondritis was the leading symptom of the disease; the patient during life expectorated nearly the whole of his

laryngeal cartilages, and I show you here the collection of these fragments, out of which at the present time one can construct a picture of the cartilaginous framework of the larynx. In connection with the prominent feature of this case, viz., the œdema, let me tell you, that whilst acute or, more rarely, chronic œdema may occur in any case of laryngeal cancer, I have seen it most frequently and most highly developed in such cases in which perichondritis has been caused by the fundamental disease—in a smaller number of cases it is not so much the cartilages as the nerves which are affected, and when branches of the superior laryngeal nerve are implicated, pain may be the predominant symptom.—More frequently the growth invades some small vessels, and the occurrence of small, oft-repeated hæmorrhages has already been mentioned as almost pathognomonic of cancer. In rare cases the disease may invade larger vessels, and then make, by sudden fatal hæmorrhage, a rapid end to the patient's life.

The duration of life in these cases varies very much. Cases have been reported of such incredible duration as ten and even fifteen years, but they are not authenticated. The longest really authenticated case I am aware of has been reported by the late Dr. Fauvel of Paris—it lasted $6\frac{1}{2}$ years. The longest case I have seen was $4\frac{1}{2}$ years; but you may regard the average duration of life in malignant disease of the larynx as between two and three years, though not a few cases occur in which the duration of life from the first symptom until death is one to two years.

The diseases with which malignant disease of the larynx is most likely to be confounded are:—(a) tuberculosis, (b) syphilis, and (c) above all others, particularly in the early stages, benign growths. Of the differential diagnosis of the latter from malignant disease I have already spoken at length, and have here only to add, that whilst in some cases an experienced laryngologist will be able, even in very early stages, to pronounce with certainty as to the innocent or malignant nature of the new growth, in others the differential diagnosis is, and may remain for a long time, the most difficult task that could possibly fall to his lot. I have myself seen a case in which it remained in doubt for over two years, the patient being meanwhile under the constant supervision of two most competent laryngologists, until

at last the malignant nature of the disease unmistakably declared itself. Similar experiences have been recorded by other trustworthy observers. Fortunately such cases are rare, and usually a two or three months' observation—the growth being *meanwhile left undisturbed by local interference*—will clear up all doubts. Of course, if the suspicious neoplasm should project sufficiently into the glottis to allow of a not too small fragment being intra-laryngeally removed and submitted to microscopic examination at the hands of a skilled pathologist, this will be the proper course to follow. Once more, however, let me emphasize the fact to you, that you must not expect impossibilities of this method of investigation. The pathologist can only give a verdict on the fragment submitted to him, and not on the whole of the affection from which the patient suffers, unless he finds in that fragment undoubted evidence of malignant disease. This he will most frequently succeed in doing in cases of squamous-celled carcinoma (epithelioma). Seeing, however, the more or less fortuitous character of intra-laryngeal operations, the fact that the piece removed in most instances will be more representative of the peripheral than of the basic parts of the new growth, and considering further that laryngeal neoplasms, like others in any other part of the body, need not be, and often are not, homogeneous in their structure, a negative histological verdict must not be allowed to overrule well-grounded clinical fears, and the decision in such cases is merely postponed; whilst, if meanwhile other grave signs of malignancy should make their appearance, the clinical observer must have the courage of his opinion, and not postpone the surgical measures, by which alone he can hope to save his patient, until he has received the sanction of the pathologist—this coming forth sometimes only at a time when the proper moment for operation has gone by.

With regard to the differential diagnosis from tuberculosis of the larynx this may be said:—Tuberculosis usually is a disease of young adult life, whereas malignant disease as I have stated is almost always met with in later life. Of course, too much reliance must not be placed on this one point, because, on the one hand, exceptional cases occur in which malignant disease has been observed at between 20 and 30 years of age, and on the

other hand, tuberculosis may and does occur at much more advanced ages than even 30 or 40. Still, on the whole, age will help you materially in diagnosing. Again, tuberculosis of the larynx is practically never a primary affection, and there is in such cases almost always concomitant disease of the lungs. Furthermore, the presence or absence of constitutional symptoms (fever, perspiration, etc.) and the examination of the sputum for bacilli may materially help you when the local appearances are doubtful. But even these cannot be absolutely trusted, because, from personal experience as well as the study of the literature of the subject, I can tell you that a few very well-authenticated cases have been observed in which there was, concomitantly, malignant disease of the larynx with tuberculosis of the lungs. One such case I observed here some years ago through the kindness of Dr. Sharkey. Post-mortem examination left no doubt whatever that there was, simultaneously, laryngeal cancer and pulmonary tuberculosis. I mention this more particularly because even now it is usually taught that there is some antagonism between the two diseases, and that cancer excludes tuberculosis. That is a mistake. Professor Hanau, of Zurich, has made a considerable collection of cases of tuberculosis and cancer in the same subject, and the array of evidence he thus brings is very valuable. There is one case recorded in which the larynx was extirpated for tuberculosis, which was considered to be cancer, but such mistakes are happily very rare.

With regard to local signs, let me remind you that cancer of the larynx always starts from one focus, whereas laryngeal tuberculosis originates infinitely more frequently in a number of different foci which gradually become confluent, and then form the characteristic tubercular ulcers. Again, the pallor of laryngeal tuberculosis is very different from the violent—often oedematous—congestion, which is seen in the neighbourhood of malignant growths. Tuberculous ulcers are at first as a rule perfectly superficial, then gradually become more and more worm-eaten in appearance, and only finally penetrate into the more deeply situated tissues. There is an absence of any such enormous, angry-looking tumefaction, limited to one part of the larynx, as is often observed in cases of laryngeal cancer, whilst considerable glandular enlargement in the cervical region hardly ever occurs in tuberculosis. Altogether, the danger of mistaking tubercular disease for malignant disease of the larynx is really not great.

Matters are different with regard to syphilis of the larynx. Even to the most experienced eye it may be almost impossible to distinguish a syphilitic infiltration from a malignant infiltration of one side of the larynx. There is an absence of definite

characteristics; the pre-formed parts of the larynx may completely disappear in the infiltration due to either disease. In the neighbourhood of both infiltrations there may be a zone of violent inflammation and oedema, and even a swelling of the cervical lymphatics will not help you very much in the diagnosis of these cases. I therefore return to the point which I have repeatedly emphasized in this lecture and in the previous one on syphilis of the larynx, viz., that in all doubtful cases, even if you are almost certain, you should give iodide of potassium at once, and in large doses. You cannot do any possible harm thereby, and you may save your patient. It is a precaution never to be neglected. Only let me warn you against trusting too quickly to the effects of iodide of potassium. Apart from being an antisyphilitic it is also a very powerful absorbent, and it may therefore have a good temporary influence in genuine cancer upon the violent infiltration and oedematous congestion in the neighbourhood of the new growth without influencing the tumour itself. The general condition of the patient may also be temporarily improved by the action of the drug upon the additional inflammatory swelling; you ought, therefore, to be on your guard against too quickly concluding that good influence has been exerted on the disease proper. Not until you see the whole tumefaction disappear under your eyes are you justified in making the diagnosis of syphilis as against malignant disease.—At a later stage also, namely, when either the malignant growth or the tumefaction of gummatous infiltration has broken down, the diagnostic difficulties may still be very great. True, we hardly ever observe in syphilis such a great tumefaction of the part as in cancer of the larynx; but you may see the laryngeal cancer at such a time that the tumefaction has already almost completely perished in the general ulceration, so that the administration of iodide of potassium is of great value in this difficult stage as well. If there are concomitant syphilitic affections, such as rupia or periostitis, or other bony affections, or ulcerations of the tongue or pharynx, or choroiditis, etc., these will help you materially in your diagnosis. Unfortunately, however, tertiary syphilis of the larynx so often occurs quite alone that these differential means are not always at our disposal. The mere presence of syphilitic scars in other parts of the body does not help you much, because even if you do find them you must never forget that syphilitic scars are centres of predilection for the development of cancer, as is often noticed in the tongue, and that malignant disease of the larynx may have developed in an old syphilitic cicatrix.

The other diseases which could possibly come into the question—such as leprosy and lupus—are such remote possibilities that I do not think it is necessary for me to discuss them here.—The same applies to gout in the larynx. I once saw a

case, in which, on thyrotomy being performed, the supposed malignant growth turned out to be a gouty deposit, and the late Sir Morell Mackenzie mentions another instance, in which he and the late Professor Krishaber mistook a gouty ulceration in the larynx for a malignant one; but such cases, evidently, are so exceedingly rare, that even in the standard works of laryngology the contingency is not mentioned.

I now come to the question of prognosis. Only a few years ago malignant disease of the larynx was looked upon as one of the most fatal diseases of mankind, and I need not tell you that even now the prognosis—under any circumstances—is extremely serious, inasmuch as even when a radical operation is possible and successful, you have always to reckon with the great possibility of recurrence. But in a considerable proportion of cases the prognosis is no longer so hopeless as it was. Our means of diagnosis have made very considerable progress, and we can now diagnose cancer at a much earlier period than previously. Partly owing to this fact, partly to improved methods of operation and after-treatment, our means of dealing with the disease by radical operation have, especially during the last few years, made really astounding progress. Quite a number of patients, operated upon by mere thyrotomy and removal of the diseased area and its neighbourhood, are now living in the best of health and free from recurrence for a considerable number of years, who, only ten years ago, would have either been left to their fate or been subjected to total extirpation of the larynx with all its risks, followed, even if successful, by the wearing for life of an artificial larynx. As to the question of voice, which is, of course, subordinate to that of life, but still important enough, the results now obtained are no less gratifying. In some cases, even after total removal of all the soft parts on one side of the larynx, the voice becomes so natural, that it is hard to believe that so radical an operation should have been carried out; in almost all others, even if somewhat weak and hoarse, still it is quite useful. A cicatricial ridge usually forms in the situation of the removed vocal cord, and the healthy cord approaching or even touching it in phonation, the glottis is almost completely closed, and a well audible voice results. Of course, as soon as both vocal cords are affected, the prognosis with regard to the voice is much worse, and for this reason again I would here once more beg of you if you have a doubtful case not to wait too long before securing, if possible, the diagnosis, and carrying out the required operation. In such cases the responsibility of the practitioner is enormous, and by allowing the favourable moment to slip by you may be justly blamed for depriving the patient of his only chance.

At the last meeting of the British Medical Association, Dr. Bryson Delavan, of New York, showed a most wonderful case of total extirpation of the larynx, performed many years ago by Dr. Solis Cohen, of Philadelphia. The patient stayed in our hospital during his sojourn in this country, and many of you may have seen him. The result obtained and the subsequent immunity from recurrence in this case no doubt rank among the great triumphs of contemporary medicine. But whilst willingly acknowledging this I feel sure that all those distinguished surgeons who do not now hesitate to perform total extirpation of the larynx will agree with me that, in the patient's interest, it is infinitely more desirable that the disease be not allowed to get to such a desperate stage as to necessitate such heroic measures, but should be, if possible, so early diagnosed that less serious interference will suffice.

The cases most suitable for radical treatment are those in which the disease appears in the form of a definite tumour on one vocal cord. In these cases either the clinical symptoms alone or with the possible help of the microscope usually will enable a diagnosis of malignancy to be made at a comparatively early period. This diagnosis having been arrived at, the operation ought at once to be performed which only about ten years ago was very much in disrepute, but which is now coming to the fore, mainly through the excellent results which my friend Mr. Butlin and I have been able to report recently. I am not quite sure about the exact proportion of successes in Mr. Butlin's operations, but in the operations of thyrotomy for malignant disease in my own practice I have been lucky enough to obtain a proportion not merely of success, but of lasting cures which formerly the best statistics did not approach, by increasing the percentage of lasting success from 21 per cent. to 58 per cent. Thus the opinion of twenty years ago that the disease was an absolutely fatal one no longer holds, provided it is taken in hand early enough and only suitable cases are operated upon. I do not go so far as to say that the operation, as improved by Mr. Butlin and myself, is no longer dangerous, but the risk to life certainly is greatly diminished.

The proceeding in these cases is as follows:—We perform tracheotomy and insert a tube surrounded by an aseptic sponge (Hahn's tube). We then wait ten minutes to allow the sponge fully to expand, and utilize this interval in laying bare that portion of the thyroid cartilage which anyhow must be split open, and which may have to be removed should the disease be found to be more advanced than could be judged from mere laryngoscopic examination. Considering that with the laryngoscope we only see the outward-growing portions of the neoplasm, and can neither accurately gauge the amount of infiltration of the

mother-soil nor inspect the subglottic cavity, you will not be surprised to hear that, after opening the larynx the disease almost always is seen to be more extensive than it had been believed to be. We then open the larynx in the median line by bone forceps, and when it has been opened put two strong silk strands through each half of the thyroid cartilage, make a knot on each side and tie, so as to have a loop. Then by drawing the two halves asunder a good view is obtained of the interior of the larynx, advantage being taken of artificial light, either by a lamp and laryngoscopic reflector, or by affixing a small electric light to your forehead. This is very necessary, so that you may be sure, on the one hand, that you are removing the whole of the malignant growth, and on the other that you remove no more than is actually necessary in order to preserve to your patient a useful voice. Having satisfied yourself as to the extent of the disease, you will find it very useful to avail yourself of a modification which I have introduced, namely, to paint the whole of your field of operation twice or thrice with a 5 per cent. solution, or even one of 10 per cent. of cocaine, and then again to wait two or three minutes. The object of this step is to obviate the capillary hæmorrhage, which formerly was a source of considerable difficulty and loss of time in operating, as it prevented the operator seeing his field of work; besides, when sponges were introduced to clear the field from blood, the cough which they produced, through irritating the peripheral ends of the superior laryngeal nerve, was most troublesome. The cocaine, however, practically disposes of that difficulty as well. Then you make—about half an inch from the growth—two semicircular cuts, which unite both anteriorly and posteriorly, and next by curved scissors you cut out the whole diseased region down to the perichondrium. Then take a sharp spoon and scrape the whole base of the wound, so as not to leave the least trace of cancerous tissue behind. In conclusion, illuminate again to make sure that you have really removed every vestige, and take any additional precautions (tying of vessels, etc.) which you may think necessary. After this dust the whole wound thoroughly with iodoform. We no longer leave the tampon-tube in for twenty-four hours, but remove it at once, and do not even put an ordinary tracheotomy-canula there from the very first. This is a very important innovation introduced by Mr. Butlin. Though I have always given every attention to having the tampon-canula quite aseptic, I have twice observed, when removing it after it had been left, according to Hahn's directions, twenty-four hours *in situ*, that it smelled horribly, and I have little doubt that it was instrumental, in one of my cases, in bringing about septic complications afterwards. After the operation we let the patient lie quite horizontal in bed, with one small pillow

only under his head, lying upon the side which has been operated upon, to allow the secretions from the wound to run out directly on to a little pad of iodoform gauze, the wound being covered with the same material. A good nurse must be in attendance, and renew this bandage as often as it becomes saturated with secretion. As to the administration of food, you usually will find that the patient will—even on the first day—be able to take a little liquid nourishment by the mouth, but you must always take the precaution of first giving him a little water from a suitable feeder. If this comes through the wound, you must feed by nutrient enemata, or by the stomach-pump. A very good plan is to let the patient lie sideways with his body partly over the side of the bed, head downwards, and let him drink from a feeder introduced into the lower-lying side of his mouth; the fluid which he drinks then completely avoids the larynx, and goes by way of the hyoid fossa, and thus no cough is produced by imbibing the food. Often the temperature does not exceed 100° the first night after operating, and very soon afterwards returns to normal, the condition of the pulse corresponding. I have had patients getting up on the fourth day after operation, and being able to leave the house on the twelfth. On the other hand, there may be very troublesome complications, notably, bronchitis and pneumonia, and the great risk of these operations consists in the occurrence—usually in the course of the first week—of these and of other septic sequelæ.

Strict antiseptic precautions and greatest cleanliness during the operation, careful avoidance of blood and secretions penetrating into the lower air-passages, unceasing attention during the first few days of convalescence are the best means to avoid these untoward complications, which, however, sometimes will occur without any apparent cause, and which are most serious in character. I have never seen a patient pull through with septic pneumonia in these cases, whilst mere bronchitic complications, though serious enough, usually take a favourable turn. Shock after the operation is another serious possibility.

The most important contra-indications against radical operation are (1) organic chest affections, (2) great old age, (3) low state of the general health. Particularly when two or three occur together, the operation should not be ventured upon.

Supposing the disease is already too advanced to allow of simple thyrotomy, either a part or half of the larynx must be extirpated. The cases most favourable for this operation are those in which the disease is situated in the front parts of the larynx, for where the growth is at the back, and the ring portion of the cricoid is involved, the patient is likely to suffer ever afterwards from serious interference with his swallowing powers, even if the

operation, which in these cases is much more serious, should be successful. Partial extirpation of the larynx practically is not attended with any greater difficulty than thyrotomy. The only thing you have to specially guard against is that when one half of the larynx is removed, hæmorrhage from the inferior thyroid artery may be plentiful and difficult to control.

Of the still larger operation of total extirpation of the larynx, I cannot speak from personal experience. There are now so many successful cases of this on record that it would be idle to speak against the performance of it, but I would repeat my solicitation to the general practitioners to see that the cases which come under their observation do not reach such a stage as to necessitate the major operation.

Finally, let me mention in this connection, that cancer of the epiglottis, or when situated on an aryteno-epiglottidean fold is more advantageously attacked by subhyoid pharyngotomy than by laryngotomy.

Unfortunately, there are still many cases which do not permit of radical operation; the patient may be seen too late, or the disease may have commenced in a totally inaccessible part. In such cases, you must be content with palliatives, and the chief of those palliatives is *early* tracheotomy. If this operation be left too late, a very usual result is the occurrence of acute bronchial catarrh, which so pulls the patient down that he practically derives no benefit from the tracheotomy. Then you may be blamed for performing the operation at so late a period. You must therefore try and convince your patient that as soon as he observes any real difficulty in breathing, tracheotomy will prolong life, and will abolish the danger of asphyxia.

In the class of cases of which I have spoken, namely, where the disease is situated on the posterior wall of the cricoid cartilage, it may ultimately become necessary to feed artificially, either by a tube, or by nourishing enemata, and even the question of gastrostomy in addition to tracheotomy may come into consideration. But I need not tell you that life under such circumstances is a very questionable gift. In such cases, your duty is to lay the matter in the gentlest form possible, but clearly, before your patient or his relatives, and let them decide. Do not take upon yourselves a responsibility which you may afterwards find difficult to bear, and which may be disastrous to your reputation.

In conclusion, I beg to say that the principal aim of my remarks has been to convince you of the urgent necessity of arriving at a diagnosis at the earliest possible moment, and then proceeding according to the dictates of your conviction without fear, and with only the one desire of being useful to your patient.

A CASE OF SUBPERIOSTEAL MASTOID ABSCESS.*

BY

J. JACKSON CLARKE, M.B. Lond., F.R.C.S.,

Assistant-Surgeon at the North-West London Hospital,
and Pathologist at St. Mary's Hospital.

OF the many possible complications of middle-ear disease, subperiosteal mastoid abscess is the most obvious, and hence the most alarming to those about the patient; it is also the most responsive to surgical treatment. The little boy to whom I ask a few minutes' attention has to-day a linear scar behind the right ear remaining from an operation done on December 7th, 1895. On the latter date he was brought to my out-patient department. The condition was recognisable as soon as the patient entered the door. The downward, outward and forward displacement of the ear in these cases renders them peculiarly easy of diagnosis. It is represented in the conventional sketch, fig. 1.

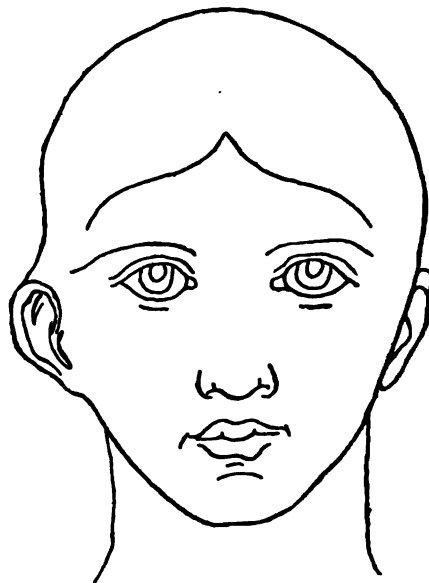


Fig. 1. Outline of a face showing the displacement of the right ear.

The history given by the mother was as follows: The child, who is 4 years of age, had scarlet fever two years ago, and ever since had suffered from a discharge of the right ear. On December 4th he complained of great pain in the right ear and seemed very ill, and remained so up to the time of admission, but he had been rather better the last day or two.

* Shown before the North-West London Clinical Society, Feb. 19th, 1896.

On examination the patient was seen to be a flabby rachitic child, with a typical subperiosteal abscess behind the right ear. The tympanum was filled up with granulation tissue, no trace of the membrane being visible. The condition demanded immediate operation, so the child was put under chloroform, and a vertical incision made down to the bone from a point over the posterior root of the zygoma, vertically along the anterior part of the mastoid process. About one ounce of pus, which smelt strongly of necrosed bone, was evacuated. A surface of bare bone as large as a penny-piece was exposed, and at the anterior

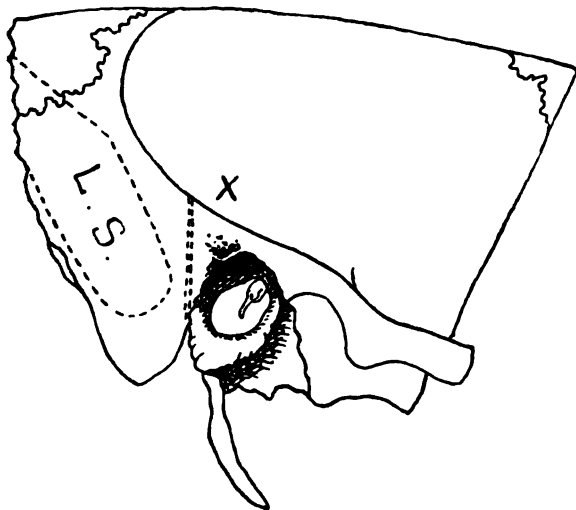


Fig. 2. The outer surface of the right temporal bone of a boy, æt. 16. In front of a perforated depression just behind the junction of the roof and posterior wall of the external meatus is the small suprameatal spine (the letter "X" is placed a little way above the spine). The double interrupted line shows the base of Macewen's suprameatal triangle: the single interrupted line corresponds with the position of the lateral sinus.

part of the exposed bone the little suprameatal spine* was evident. A glance at fig. 2 may facilitate the references to the anatomy of the part.

The region immediately behind this spine is the outer wall of the mastoid antrum. Macewen has more narrowly delimited the area of bone to which the excavation is to be limited. A line drawn vertically upwards from the postero-superior extremity of the tympanic bone to the posterior root of the zygoma, forms, roughly, with the posterior border of the meatus and the root of the zygoma, a small triangle, of which the suprameatal spine is the apex. In the case under consideration, a small gouge was used to remove the outer wall of the antrum, which was about $\frac{1}{2}$ in. in thickness. The antrum was cleared out. It contained small,

firm, polypoid granulations resembling those found in suppurative disease of the frontal sinuses. The question now arose as to whether it was desirable to join the antrum and the tympanum, clearing out the granulations and the remains of the malleus and incus. In a former paper* I have stated the opinion that in children, in uncomplicated cases of subperiosteal abscess, it is sufficient to open and clear out the antrum thoroughly—usually only a short operation, whereas that of joining the antrum and the tympanum requires a considerable time—about half an hour. The opinion was chiefly based on a consideration of the mutual relations of the two cavities. This is shown in fig. 3, which

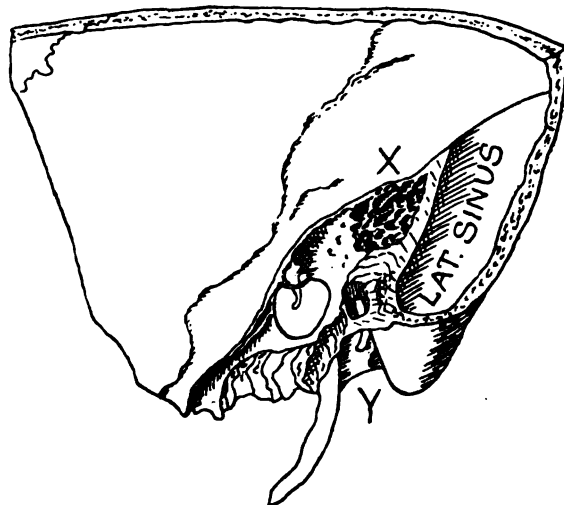


Fig. 3. Inner aspect of a temporal bone from which the petrous part has been removed. Below "X" is the mastoid antrum, above "Y" is a portion of the aqueduct of Fallopius and the facial nerve.

represents the inner surface of the preparation shown from the outer side in fig. 2. It will be observed that the roof of the antrum is continuous with that of the tympanum, and is inclined at an angle of about 45° with the horizontal. The floor of the antrum, on the contrary, is on the same level as the roof of the tympanum, so that the antrum has a drain-way through the natural communication between the two cavities. My opinion has been confirmed by the results of an autopsy I was lately privileged to make for Mr. Ernest Lane, who had performed the mastoid operation three years before the patient died from another cause. The disease had been completely cured. In fig. 4 is shown the upper half of a section made in an oblique plane, so as to pass through the middle of both antrum and tympanum. The enlarged antrum, which was closed externally by a fibrous membrane, was free from pus, and the tympanum, though only a fraction of the drum-membrane remained, was normal in other respects.

* The usefulness of this little landmark is shown in an article by the Author in the "Journal of Anatomy and Physiology," vol. xxvii, p. 411.

* *Lancet*, July 14th, 1894.

In the case I am now considering, the presence of granulation tissue in the tympanum and the strong smell of dead bone made it necessary to open the antro-tympanic isthmus. This was done by beginning at the depression behind the suprameatal spine, and working along the roof rather than the posterior wall of the meatus, until the notch of Rivini was reached. The dangers run in this

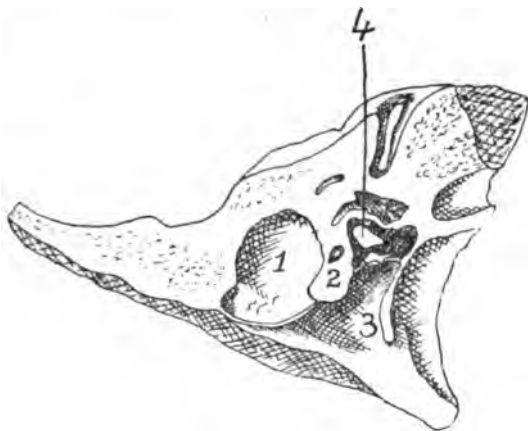


Fig. 4. The upper half of a section of a temporal bone in which the antrum has been opened.

1. The dilated antrum. 2. The posterior wall of the meatus, containing the aqueduct of Fallopius and (deeper) the external semicircular canal. 3. Marks the entrance of the ext. meatus. 4. Shows the normal communication between the antrum and the tympanum.

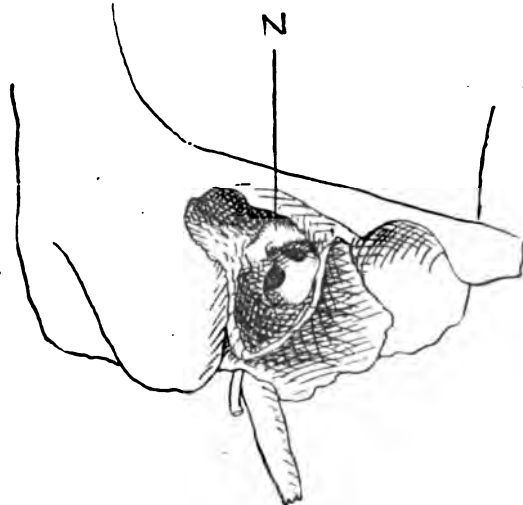


Fig. 5. A temporal bone in which the antrum has been opened and thrown into the tympanum. The guideline "N" marks the aqueduct of Fallopius. The posterior wall of the meatus has been cut away lower down than is done in operating.

proceeding are well known. In fig. 4, in the bony septum between the two cavities, are seen the facial nerve and the external semicircular canal, cut across in a section which passes through the middle of the posterior wall of the bony meatus. Fig. 5

shows a preparation made with a dental bur to show the position of the aqueduct of Fallopius. The importance of keeping close to the roof of the meatus will be evident. The operation was completed by removing the remains of the malleus and incus, and the granulations from the tympanum, and, finally, a careful examination of the roof of the cavities and of the posterior wall of the antrum for any communication with the extradural space or the brain. Nothing being found, the cavities were lightly packed with gauze. Convalescence was marked by one troublesome feature, viz., the onset of facial paralysis some thirty hours after the operation. I carefully examined the patient for this symptom before the operation, and again when the child had recovered from the anæsthetic. There was no trace of it on either occasion, but about thirty-six hours afterwards the house-surgeon, Mr. Stroud Hosford, noticed partial paralysis, which became complete in a day or two. Three weeks after the operation the paralysis had completely passed away. It was, I think, caused by œdema within the aqueduct. The opening into the antrum was prevented from healing too rapidly by daily packing with gauze. Five weeks after the operation the opening had closed, and the discharge had ceased. The patient then heard the watch on light contact with the pinna. At the present time he hears the watch at a distance of 1 in. from the ear. For at least two months longer regular syringing will be practised, and after that the ear should be inspected occasionally, and not less important will it be to watch the condition of the left ear, and to remove thoroughly some adenoids which are present in the naso-pharynx.

One word I would like to add before closing. It is often said that so long as a discharge is abundant there need be no anxiety about a case of suppurative ear disease. There could hardly be a greater mistake—a mistake which has cost many valued lives. I would say that if, in a case of chronic suppuration in the tympanum, regular syringing, combined with Politzerisation, fails to cure the discharge after adenoids or polypi have been looked for, and removed if found, the chances are that the pus comes from the antrum, and that a prompt operation on that cavity may prevent the supervention of extradural or cerebral abscess, sinus phlebitis, or some other of the graver complications of middle ear disease.* In every case of obstinate middle ear disease the pus should be carefully examined for tubercle bacilli, and minute inquiry made to ascertain whether the patient has suffered from syphilis. Tubercle or syphilis will modify the prognosis unfavourably.

* The discharge may even come from the interior of a cerebral abscess. The author has described and figured (Field, "Diseases of the Ear," fig. 70) a case in which the discharge came from a cerebellar abscess which communicated with a dilated antrum, and the latter again with the tympanum.

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 4, 1896.

A LECTURE

ON

TRAUMATIC NEURASTHENIA.

Delivered at University College Hospital, February 10, 1896.

By VICTOR HORSLEY, F.R.S., F.R.C.S.

GENTLEMEN—The case of Traumatic Neurasthenia I shall demonstrate to you this afternoon is of such an interesting character, and presents so many points of the greatest practical importance, that I considered it worth while to deliver a lecture on the case. To put the position roughly, it is the case of a man who has injured his back by a fall. There are two headings under which this lecture may be divided—first, the clinical interest and importance of the treatment of such conditions, and, secondly, the medico-legal treatment—if I may use the expression—and the consideration of which will be deferred until after the patient has left the theatre.

The clinical aspect of the case and its treatment will first occupy our attention; and to begin with, we must look to the man's previous history. The patient has had scarlet fever and small-pox, is 53 years of age, and his family history is good. On the 12th of December he fell from a scaffolding on to a heap of bricks, a distance of fourteen feet, falling on "the small" of his back. Upon admission into this hospital he complained of severe pain in his back reaching down to his thighs, the pain being most acute on the right side, and upon examination there was found tenderness over the twelfth dorsal and the first lumbar vertebræ. There was no loss of motion or sensation, though the knee-jerks were absent, the urine was passed normally, and no injury was to be detected in the pelvis. This summarises briefly the acute condition of the case; he continued to have considerable pain, and on the third day his temperature, which had been raised from the evening of the accident, rose to 104° , remained up for four days, and then fell. Concurrently with this

condition of temperature the following physical signs were noted in the chest—dulness on the left side, and later on friction sounds were also heard on the same side, and a very violent cough set in which caused him great pain in the back, and produced an expectoration not only purulent, but also rusty, its rusty character being noted for several days. It is quite clear, therefore, that he had some visceral trouble apart from the injury to the spine and central nervous system. These facts at the onset of the case, should lead us to divide the examination of such a patient in the following way. First, of course, we examine his back, the injured part; secondly, the condition of his nervous system, his brain and spinal cord; since it is not to be supposed that anyone can fall fourteen feet without some symptoms of concussion, meaning thereby concussion of brain rather than spinal cord; thirdly, we examine the state of the viscera generally. Careful investigation under these three headings should constitute the systematic examination of such a case when first coming under our notice. On looking further into the notes, the present case soon simplifies itself into pain in the back and down the leg as the chief complaint, with a gradual subsidence of the chest symptoms; one point more, however, and that a most important one remaining to be noted, as follows:—on the 22nd of January (the accident was on the 12th of December), the patient sat up for two hours in a chair, and after getting back into bed again he gave a cry and had a fit, remaining unconscious for five minutes, the teeth were clenched, and the urine and fæces were not passed. Directly after the fit the patient was quite sensible again. The fact must be clearly borne in mind, that though the pain in the back had practically gone, yet when the patient began to move about he had a fit.

What, now, is the present state of this patient? To answer this the condition of the spine must be first determined, and in this physical examination of the spine we lay the patient first on his side. In this case we may be tempted to say that

the dorso-lumbar region is more prominent than normal. On examining the individual spines in that part, by pressing them from side to side, nothing abnormal can be detected, no thickening round them, nor any looseness, nor, as is sometimes found, any crepitus in the neighbourhood, not from a fracture of the spine detaching it from the laminæ, but from a small portion being torn off by its tendinous attachment, this being the best proof of actual injury to the spine. At the last annual meeting of the British Medical Association I showed a case of a man who had fallen off a cart and fractured his spine, and yet upon examination nothing at all abnormal could be detected except a little sharp piece of bone torn off one of the spinous processes, though when laminectomy was performed it was found that the body of the sixth cervical vertebra was fractured. Externally there was nothing to indicate the extent of the mischief except the small fragment of bone torn off at the time of the accident. Upon placing this man face downwards upon the couch, the lumbar region still looks abnormal, for instead of the usual slight hollow, distinct prominence is its salient feature. The next thing to do is to examine the man sitting up, and still these two vertebræ, the twelfth dorsal and the first lumbar, stand out more prominently than natural.

Normally, in an adult, these spines, as a whole, become more prominent upon the sitting posture being assumed, and in some few people it may happen that only two or three spines become prominent; but in this particular patient these two spines are so prominent that they most certainly suggest something more than mere individual anatomical peculiarity. When pressure is applied to the man's shoulders, his finger is drawn over the left lumbar region to indicate where the pain is felt, showing that the pain is limited to this area, that it does not come round to the front of the trunk, and that it does not follow the distribution of any of the known nerves, but, in the main, seems to be an affection of some of the posterior divisions. Upon the man bracing himself up, when the spine is naturally thrown forwards, the prominence in the lumbar region does not completely disappear. So far as we have gone, the conclusion arrived at is, that certain vertebræ are more prominent than usual, and that there is a patch of pain to the left side of the pelvis which,

however, does not come forward into the hypogastric region. The next step in our procedure is to make the man lie upon his back, and we now examine his legs and inquire into the condition of the cutaneous sensibility which we find to be normal for all forms of stimulation.

As regards the reflexes the superficial are normal, but as regards the deep reflexes it must be understood that his knee-jerks were absent on his admission to the hospital; and although, of course, we do not know that they were present before the accident, yet it is, at least, reasonable to assume that they were, always remembering that in this, as in all cases, there may still be some error in observation. At the present time there is a brisk jerk on the left hand side, but it is absent on the right. The man complains of pain down the front of the leg, and on my requesting him to indicate the position by pointing with his finger, it is to be noticed that the area described is over the course of the external cutaneous nerve. Having arrived thus far, I think it is now convenient to send the patient back to his ward.

Now to consider the possible localisation of the injury. If we were to remove the spines and laminæ of the twelfth dorsal and first lumbar vertebræ then we should find ourselves opposite the lower end of the spinal cord and of the nerve roots, the first coming from the cord here would not be the last dorsal or the first lumbar, but almost directly opposite the middle of the laminæ would be the third lumbar, so that if there was any point where the nerve roots were injured, the roots of the first and second lumbar nerves would be most likely to be damaged by the accident, and any hæmorrhage would be amongst these roots.

As a matter of fact, this patient now presents these symptoms only—pain in the back on movement, and pain in the distribution of the external cutaneous nerve on both sides. Inasmuch as on the left side the knee-jerk is brisk, and there is no bladder or rectal trouble, if this patient has any organic mischief at all, it would be injury of the vertebræ mentioned and of the nerve roots (possibly hæmorrhage or bruising). The man in addition, of course, suffered from the chest complication already described, and he was ill altogether for a considerable time, and this, added to the fact that he fell a considerable height, viz., fourteen feet, in consequence of which he suffered from shock, has

brought him into what used to be spoken of as the traumatic condition, and which should be spoken of as traumatic neurasthenia. Now, what will become of a case like this? It is probable that this pain in the back will continue for some time, and it is also probable that when he goes out of the hospital and when he goes back to work he may find the pain still persists, and even gets worse, and also the more he may try to do, the worse will the pain get. Now it is quite in accordance with human nature that when a patient thus finds himself getting no better, he should begin to think who is responsible for his accident, and then it occurs to him that the responsibility rests on the employer, whereupon he naturally finds out whether his employer will compensate him. In this way, a number of cases will come under your care, cases of what is called traumatic neurasthenia, and these cases are most important. They are of the utmost importance to us as medical men because they frequently find their way into the law courts, and then you may find yourself called upon to go into the witness-box, and say precisely why the patient has not got well, and you will find it no easy task to explain to a jury what is the matter, or what the present result is due to, or what was the condition at the time of the accident.

This naturally brings us to the medico-legal part of this subject. These traumatic neurasthenia patients are very much in the same condition as those patients who have suffered in a railway accident, who, not actually having been wounded, nevertheless, some weeks after the accident, begin to have pain in the back, and then to develop other symptoms similar to those of this patient. The array of symptoms they so characteristically present were first described by Mr. Erichsen under the term "railway spine." The greatest authority on so-called hysteria and neurasthenia is Charcot, who pointed out that in the vast majority of cases of traumatic neurasthenia where the patient has been subjected to an accident, and where, because he felt no ill-effects, at once thought that he had escaped without any injury. Such a patient, nevertheless, passed through a kind of incubation period, most commonly a week, sometimes three, and would then present certain symptoms—symptoms which this man we have seen to-day has presented throughout his case. Speaking from a considerable experience of these cases, I notice that this curious

sort of pain along the distribution of the external cutaneous nerve is an extremely common symptom. I am quite unable to explain why it occurs so frequently. In this particular case we have made out that possibly certain nerve roots were injured; but, to put you on your guard, I will tell you of a similar case arising out of a railway accident, in which a man was thrown violently forward, and struck the opposite seat with his abdomen. This man complained, exactly like our patient, of pain in the back, and pain down the outsides of his thighs; he had other symptoms, too, which led me to diagnose neurasthenia, but I could not make out any organic injury to nerve roots, and it was my opinion that under the Weir Mitchell treatment he would get quite well. However, for various reasons he did not go through this treatment, and ultimately he brought an action against the railway company. The medical officers representing this company felt themselves justified in diagnosing hæmorrhage into the last dorsal nerve—a somewhat recondite diagnosis to make some months after an accident. At any rate, the man got the benefit of the doubt, and received £3,500, without the case going into court at all. I need hardly say that within a few weeks he was perfectly well, and walking about.

These cases must be investigated by you on some systematic method, and first, we will take the possible changes set up by the injury in their higher nerve centres. These patients, even when they have been genuinely injured, and are in real suffering, are never reliable in their statements. It is no evidence, therefore, of malingering if you find some contradictions in their statements. Further, they are emotional and apprehensive, they cannot sleep, and when they do sleep they have nightmare, and their so-called volition or voluntary power is greatly diminished, they very often honestly desire to attend to their business affairs, but they cannot do so. These symptoms are all *subjective*; and though you have to form your judgment on such statements, they will be naturally discounted by the jury. The next point to consider is the condition of the lower centres. All these patients say, in regard to their power of movement, that they cannot walk half a mile without exhaustion, and that statement is perfectly true, for they cannot voluntarily move more than that, they are not paralysed, their "motor centres"

become exhausted. Under certain circumstances you will find that if they happen to have had any slight injury to one of their limbs, that not infrequently that particular limb will become useless, and, though this is only a functional monoplegia which will disappear under appropriate treatment, still the fact remains that it is a reality to the patient. You will not find on examining these patients any objective signs of loss of associated movement, such as, for instance, occurs in diplopia from obvious paralysis of one of the muscles of the eyeball. Next, as regards sensation, all these patients suffer to a certain extent from varying alterations of sensation. With regard to the special senses the sense of smell and taste is not interfered with. You will remember that in many cases of organic brain disease, smell and taste disappear. In reference to the sight it is the commonest experience to examine the eye, and find no objective change; and yet these patients say that their sight has become worse, that the vision is misty, but that on a "good day" as it is termed, their sight is clear. When much general pain in the head is complained of, the vision is usually described as misty, and it is no doubt an actual statement of the fact. This weakness in the sight is probably a central phenomenon, *i.e.*, fatigue of the visual centres of the cortex. There is a rare condition which must not be forgotten, and which should always be looked for in these cases, and that is hemianopsia; these patients may complain that their sight is not equal at seeing things from one side, and you may often find that even if they have not hemianopsia, they may have contraction of the field of vision, and sometimes a certain degree of colour blindness. The hemianopsia is of great importance, and within my own experience I have come across cases in which the hemianopsia was discovered before the case came before the law courts, and in which it has been said at the trial by the medical men even on the plaintiff's side that it would probably disappear, but if you read Charcot's great work on "hysteria" you will find that he looks on hemianopsia so far as prognosis is concerned as a very serious symptom indeed. In one of the cases I have just spoken of, and in which the accident occurred seven years ago, the hemianopsia still persists as clearly marked to-day as it was then. It is therefore a very serious symptom, and it cannot be discounted in quite the same way as these

other symptoms can, of which I have been speaking to you.

As to hearing, it is very common for these patients to say that they have a buzzing in the ear, in the same way as an anæmic patient will complain of buzzing noises. Also dizziness is frequently mentioned; but on careful inquiry you will discover this to be not true vertigo, but a subjective sensation of "swimming in the head." With reference to common sensation it is rare for these patients to have true anæsthesia. Of course, you know that in "hysteria" tactile æsthesia is frequently diminished, and even analgesia may be present; but in traumatic neurasthenia cases it is unusual, though there is one thing in common between these two conditions, *viz.*, very often a certain degree of loss of muscular sense. Passing from sensation, we come to the state of the reflexes, the superficial reflexes are usually unaltered, but the deep are most commonly exaggerated, and if the condition is not relieved by treatment they may disappear. I was once taken to task in the high court by an eminent barrister, who asked me what the condition of reflexes were in a certain case, and I stated that at first the knee-jerks were exaggerated, but that when I examined the patient subsequently (having had the opportunity just before the trial) the knee-jerks had practically disappeared. He attacked me as to the possibility of that being so, which only shows how difficult it is for the legal mind to understand medical matters. The fact being that, in this particular case, the patient had simply got worse and worse, not having been properly treated.

As regards the various functions, the so-called visceral functions of the central nervous system, and which contrasted with those I have discussed, are more objective ones, the vesical and rectal are most important, because it is almost always the lumbar or sacral region that is injured generally most directly. What you usually find is that a number of these patients complain of frequency of micturition. Frequency of micturition is a well-known nervous phenomenon brought on by emotion, and there is nothing surprising in that; but a much more interesting fact is that these patients have nervous polyuria, that is to say, they secrete a very large quantity of urine of a very low specific gravity, and very slightly coloured. This is, of

course, a purely functional symptom, but is by some lawyers looked on as an important point, though of course it is not a whit more important than the other factors of the case, but because it is something you can tell a jury it has acquired a fictitious importance, and in courts of law I have heard this symptom made a great deal of. In these cases the rectum, you may take it, is practically never impaired, not even in the very worst cases I have seen, including cases of motor and sensory paralysis.

Passing from this review of the symptoms discovered on examination, we come next to the question, how are we to tell the difference between traumatic neurasthenia and malingering? because, obviously, as medical men we must be able to detect malingering, otherwise we shall be compounding felonies, in helping men to obtain money under false pretences. This is just where the matter is most difficult, and it is just where a medical man is apt, in his sympathy for his patient, who is undoubtedly injured, to go further than he is absolutely warranted, in giving his evidence. In giving evidence, all you have to do before going into court is, to review the case, and if there is nothing objective, you are warranted in saying that if the patient is treated by the Weir Mitchell process he will improve, at the end of about six weeks or so his main symptoms ought to disappear, and in six months he ought to be fit to return to work. Having then made up your mind as to the probabilities, all you have to do on going into the witness-box is to recount the symptoms which are abnormal, to give your opinion of the probable prognosis, and what ought, in your judgment, to be the treatment, but beyond this you ought not to go.

If you go further a clever counsel will invite you to give your opinion on the whole range of traumatic neurasthenia, though you may not have seen more than ten or twelve cases in your career, and certainly not more than thirty or forty. If you are not in a position to give your opinion as an authority you can always avoid giving opinions of an expert character, because you are only called upon for evidence on the points I have named. Not unfrequently, however, one sees a medical man stating that he thinks the patient is very seriously injured indeed, and yet when he is cross-examined as to why he thinks the patient is so

seriously injured, he finds considerable difficulty in giving precise reasons for his opinion, and thereupon the judge and the jury discount the honesty of the witness and are inclined to take a very severe view of the *bona fides* of the medical man, whereas he was really acting in good faith, and it was only his natural sympathy for his patient which led him into the region of surmise rather than fact.

Finally, I come to the discussion of the treatment of cases of traumatic neurasthenia, and provided you are satisfied that there is no real injury to the spine, the treatment resolves itself into the adoption of the Weir Mitchell method as the only reasonable plan. Remember that it is useless to simply send such patients away for a voyage, or into the country. As a rule they do not get any better, the pain in the back remains just the same, and if they are honest they are only worrying all the time to get back to their work, while you in the meantime have left undone that building up of the nervous system without which they cannot quickly get well. I have seen patients sent abroad for three months, and come back as bad as they went, it being believed that rest alone would effect a cure. If, besides, they have brought an action against any one, it stands to reason that their nervous system will be in a state of irritation till their case is settled, which is an easily understood condition.

The Weir Mitchell treatment is so limited in the minds of many as a method to be recommended solely in cases of hysteria, that it has, perhaps, rather fallen in some quarters out of use for the many conditions to which it is applicable. It is a very narrow view to take of Dr. Weir Mitchell's discovery, because it can be applied to persons who are genuinely injured, with a fairly certain expectation of recovery. What then are the essential principles of this method. Firstly, the patient must be absolutely isolated. You mean by that, that he must be put in a room by himself and seen only by his nurse and medical attendant, receiving no letters and writing none, seeing no friends, and stopping in that room for at the very least six weeks; if a mild case, say one month. While thus isolated he is put on a particular diet, first he has half a pint of milk and a rusk every two hours, till his stomach gradually becomes accustomed to taking a great

quantity of fatty matter, which given to him at once might bring on indigestion; on the third day he is put on full diet in addition to the milk, so that at the end of the week he is taking the ordinary diet with the milk in addition, and if he suffers from indigestion a couple of grains of calomel and some pepsin soon relieves him of that trouble. Isolation, then, is the first principle, over-feeding is the second, while, thirdly, there is the rubbing, and in that there is no mystery at all, he is simply rubbed all over the body, and, in addition, these patients do better if, in the third week, you add hot douches to the spine, with the water as hot as the patient can bear it, at a temperature, say, of 100° to 105° . Then the faradic current is of great assistance, though naturally no specific advantage accrues from the use of this current. Unfortunately, the excess of human nature in the public leads them to see some wonderful virtue in electricity, and they think it is a specific form of cure, whereas its only value is it improves the strength of the erector spinæ muscles. It is, however, no small point to relieve pain by the current, and in lateral curvature brought on by weakness of the muscles, the pain absolutely disappears if the muscles are treated by faradism and gymnastics. In these cases you also gain by treating, in the same way, the muscles which have more or less wasted.

A few more words are necessary concerning the fit, which the patient, whose case is quoted at the beginning of this lecture, exhibited on January 22nd. How comes it that a man of 53 should have a fit some six weeks after an accident? The character of the fit, from the brief account in the notes amounts to this, that he had a slight idiopathic fit; all four limbs were engaged in clonic convulsions, there was a slight cry, but no passage of urine, and the tongue was not bitten, but there was complete unconsciousness. The fit then was a generalized convulsion of a very low type. Brown Sequard has shown that if the spinal cord be injured by crushing or concussion in a guinea-pig that fits of this character occur in the animal just in the same way as it occurs after injury of the sciatic nerve. One is always on the look out for anomalous cases, and I have had two cases, in one of which I felt bound to do laminectomy in which fits followed an accident.

In one case it followed after a year, but in the other after a few weeks—seven or eight weeks. In this case we are considering to-day, it occurred five or six weeks after the accident. It is therefore clear from experiment, and from clinical facts that it is quite possible to have a patient exhibiting a fit, the cause of which may be an injury to the spine, or possibly to the nerve roots, and I suppose this case we have considered to-day to be a case in point. The fit being due to the accident gives a much more serious aspect to the case, and makes the prognosis much more unfavourable.

ANOMALIES OF THE TESTIS IN RELATION TO HERNIA.

Illustrated by Cases.*

By W. McADAM ECCLES, M.S. Lond.,
F.R.C.S. Eng.

Assistant-Surgeon to the West London Hospital and to the City of London Truss Society, Surgeon to the St. Marylebone General Dispensary, etc.

GENTLEMEN—The anomalies of the Testis, with which we have to deal to-day, are conveniently grouped under three headings:—

- (1) Complete descent of the Testis, but failure, wholly or in part, of the subsequent obliterative changes in the processus vaginalis.
- (2) Arrest of the Testis during its transit into the scrotum.
- (3) A combination of these two conditions.

It will be my endeavour to explain as clearly as possible, and to illustrate by cases, some or all of the ways in which these anomalies may be associated with hernia.

(1) The processus vaginalis is that finger-like process of peritoneum which precedes the testis into the scrotum, and has at first its cavity in direct communication with that of the abdomen. When the testis, however, has reached its normal position at the lower part of the scrotum, this process immediately tends to become converted

* A Post Graduate Demonstration given in connection with the West London Post Graduate Course on Feb. 19th, 1896.

into a fibrous cord, except that part which remains as the tunica vaginalis testis.

The obliteration, which usually begins at the internal ring, may be arrested before it is complete, thus leaving the funicular portion of the processus vaginalis patent; or the closure may never occur in any part, so allowing a free opening into the peritoneal cavity to remain. Now this partial or completely unclosed canal, more often found on the right side than the left, is by no means uncommon even in the adult, and still more so in children; and yet it would seem that only a minority of persons who have this peculiarity are the subjects of hernia.

This fact, I think, tends in a forcible manner to prove that it requires more than a patent sac of

its centre, indicates a strong probability of its being of the congenital form. Sometimes, however, the testis is quite at the bottom of the sac.

(2) The Testis, when arrested in its descent, may be found as indicated in the table.

It is not our intention to enter on any discussion as to the causes, complications, and treatment of such anomalies, except in so far as they are related to hernia.

All varieties of retained or misplaced testis may be associated with rupture; but when the testis lies in the inguinal canal, there is the greatest tendency for the production of a hernia, and that, practically, always of a congenital variety. There is no doubt that arrest of the testis, after it has

Anomalies
in
Migration
of
Testis.

Testis
undescended
into
scrotum.

Testis
descended but
inverted.

Arrested at some point in its *normal*
course = Retention.

- (a) In abdomen.
- (b) In iliac fossa.
- (c) In inguinal canal.
- (d) In cruro-scrotal fold.

Arrested at some point *outside* its normal
course = Ectopia.

- (a) In the perineum.
- (b) In Scarpa's triangle.
- (c) At root of the penis.

peritoneum to cause protrusion of viscera. The open mouth of the processus vaginalis or its funicular portion may in some cases, and that even in adults, be associated with the sudden appearance of a hernial protrusion on some strong expulsive effort. For it is hardly likely that the normal peritoneum covering the internal ring can be sufficiently stretched in so short a space of time as some herniæ seem to be produced in.

Strangulation of these so-called congenital herniæ is very frequent, and, when present, usually extremely acute. The diagnosis of a protrusion into the processus vaginalis, if it be scrotal is fairly easy, but if into the funicular portion only, it is almost impossible without dissection. Given a scrotal hernia, the envelopment of the testis by the contents of the sac, and the organ being found at the posterior part of the swelling, a little below

passed the internal abdominal ring, does create a predisposition to inguinal hernia, and that, perforce, on the side where retention is present.

It is difficult, however, to say that inguinal inclusion must necessarily be a precursor of hernia, for the patients who present themselves without any rupture may or may not in the future be subjects of it.

The tendency to hernia is dependent upon two factors; firstly, the very common occurrence of a patent process of peritoneum which may continue so throughout life, and, secondly, that non-descent of the testis is apparently in many cases associated with a weakness of the abdominal walls. In early life this markedly frequent occurrence of arrest of the testis, combined with hernia, is well shown by the fact that 7·5 per cent. of all scrotal ruptures begin in the first year of life, but 35 per cent. of

those ruptures in patients with included testes appear during the twelve months following birth.

If one carefully looks into a series of cases of abdominal and inguinal inclusion of the testis, it will be found that the former variety appears to be distinctly more often accompanied by hernia appearing early than the latter. It might be thought that it could be more likely that the comparison between these two sets of cases would reveal an exactly opposite result, for should not abdominal retention have probably a smaller and narrower process of peritoneum into which a rupture may protrude than in the case where the testis has actually passed into the canal? But statistics will force the conclusion that the more complete the arrest, the greater is the number of herniæ in subjects of a tender age.

I will here show you a man, æt. 49, who is the subject of abdominal inclusion of the left testis, and on careful palpation I can find no evidence of its existence. His right testis, in addition, is small. He is married, and has had children. He presents, moreover, a right inguinal hernia, as well as a marked double femoral protrusion. I do not think any one of these three herniæ are dependent upon the existence of the included testis.

The next case is a man, æt. 23, who has also abdominal retention of a testis, namely, the right; but by delicate manipulation you can feel a small body in the right iliac fossa which slips away under the examining finger, and which, he says, gives him some testicular sensation.

It will be noticed how completely undeveloped the right half of his scrotum is. He has also an inguinal hernia, but again it is on the side opposite to that of the inclusion of the testis, and it is impossible again to say whether there is any association between the two conditions.

I have another case to bring before you, a man who exhibits his right testis in the inguinal canal, and associated with this anomaly a condition known as an interstitial hernia.

Such herniæ are very interesting, especially in relation to undescended testis. It may be said that an interstitial hernia is an inguinal hernia, which, having come into the inguinal canal, does not, in the majority of cases, at least, escape at the external abdominal ring, but passes between the planes of the abdominal wall outside the canal. Certain of these are accompanied by a

ventral swelling, and as these are the cases which commonly have partially descended testes, I shall allude only to them.

It is possible, perhaps probable, that the occurrence of a testis in the canal may prevent the protrusion of the hernia through the external ring, and favour its mounting up between the layers of the abdominal wall.

The small ill-developed external ring in females may tend towards the same result. Interstitial hernia is certainly more common on the right side, and, similarly, right testicular inclusion is more frequent than left. Frequently, in such cases, a band of the gubernaculum testis can be traced down into the scrotum from the canal.

These interstitial herniæ sometimes assume very large proportions; but the case I show you to-night is a very moderate one, yet exhibits well the characteristics of such protrusions—an oval tumour, parallel with Poupart's ligament, and tending to enlarge upwards when the patient coughs.

The next case is one where the left testis is in the canal, and smaller than usual, but still with definite testicular sensation. This young man has a left inguinal hernia.

Further, here is a man, æt. 18, who has his right testis at the mouth of the external ring, that is, just outside the aperture. He is also the subject of a left inguinal hernia.

I now show you a very common condition, namely, a testis high up in the scrotum, but with no tendency to descend, thus proving that it is not a case of mere retraction, but one of true arrested descent. This patient presents, curiously enough, a right femoral hernia and a left inguinal, although it is the right testis that hangs high.

My next case is a rare and very interesting one—a man, who has a testis which has made for itself, as it were, a pocket in the cruro-scrotal fold. You will observe that the right half of the scrotum is undeveloped, and although there is a large hernial protrusion it still presents its rugose appearance. The rupture has, like the testis, descended into the cruro-scrotal fold.

Again, here is a man who has his right testis, somewhat smaller than normal, in the perineum. It has distinct testicular sensation. A well-marked gubernacular band passes from it to the tuber ischii. He had a right inguinal hernia when

an infant, for which he wore a truss, but now he shows but little evidence of protrusion. I have to thank Mr. C. B. Lockwood for kindly sending me this patient to-day.

CLINICAL LECTURE

ON

ASCITES.

BY

HENRY WALDO, M.D., M.R.C.P.,

Physician to the Bristol Royal Infirmary.

GENTLEMEN,—The case I have selected for my lecture to-day is that of a little girl, *æt* 9, in the children's ward. Her friends sought admission for her on account of abdominal dropsy. The health of her parents had been undermined by alcohol. The child's health has been fairly good till 15 months ago, when she had a cold and cough, and occasionally vomited. Last April, she was sent back from school with all the signs of a vomica in the apex of her right lung. Some months after this her abdomen began to swell, and her liver was first noticed to be very much enlarged, extending beyond the umbilicus. It was apparently smooth, and its edge felt to be thin and sharp. The temperature has been somewhat raised from time to time for a few days together, but has been mostly normal. The heart sounds are clear. The veins are full, especially those in the thin abdominal wall, and there is considerable duskiness of the face and lips, this varies very much, sometimes the lips being quite purple. Pulse small, regular and frequent. There has been an absence of jaundice; she has a chronic cough, which is more troublesome at night, with very little expectoration. I have examined this for tubercular bacilli upon two or three occasions without finding any. The voice is quite clear. Wasting is a very marked symptom, being quite general, and obliging the patient to be upon a water-bed, although she gets up upon most days. Tongue clean, bowels quite regular. Appetite very good. Although the liver is so much enlarged I have never been able to feel the spleen. The urine is exceedingly scanty, no blood or albumen or sugar present. There is no

doubt about the ascites as we have tapped the abdomen eleven or twelve times, and each time brought away a large accumulation of clear serum (the amount varying from 142 oz. to 78 oz.), but the etiology of this in conjunction with so large a liver is rather obscure. You will notice that the umbilicus is distended, and that the abdomen is resonant in front when the patient is lying upon her back. When the abdomen is very much distended with free fluid it may be dull owing to the bowels being unable to reach the surface.

The same thing occurs sometimes when only a moderate quantity of fluid is present from the mesentery being in a condition of chronic inflammation, and so contracted in consequence, that it tethers the bowel and keeps it away from the surface. So that this resonance in front in this case is rather opposed to its being one of tubercular peritonitis, although there are the signs of a vomica in one lung, and much wasting. Neither has there been felt any thickening of the omentum, which in these cases become indurated and nodulated. By shifting the patient you can demonstrate that the fluid is free, and not cystic. Fluctuation is well marked, the thrill being easily produced and conveyed all over the abdomen. The large liver is at once felt by "dipping," in which a certain part of the fluid is pushed aside, and one comes suddenly down upon the organ. Upon two or three occasions during this child's illness, there have been symptoms of drowsiness almost amounting to coma. These cerebral symptoms have often been observed in ascites, and especially after tapping. That they do not always depend upon jaundice is well known, and there has been no jaundice in this case.

The signs of vomica in the apex of the right lung are deficient expansion, with a little flattening, a dull percussion note, cavernous breathing, increased vocal resonance, with whispering pectoriloquy. It is well to remind you, perhaps, that these signs, although often associated with a lung cavity, may be present with a solid piece of lung without any excavation. The cause of the ascites is not at all clear. It is unlikely to be a part of a general cardiac dropsy, as there is no audible murmur or other signs of valvular defect. The same applies to renal dropsy, as there is an absence of albumen in the urine. It may be the result of inflammation of the peritoneum, perhaps

tubercular, with a large fatty liver—this is a very plausible theory with a lung disease. There is, however, no hardened omentum to be felt, neither has there been effusion into either pleural cavity, and the skin is usually dry. Then, again, there has been a total absence of any sign of enteritis. A fatty liver, too, is never so large as this one appears to be. It is not to be explained upon the supposition of lardaceous disease or amyloid degeneration of the liver, I think, as the spleen and kidneys do not at any rate participate, and it is doubtful if ascites occurs from a lardaceous liver. Neither is it likely to depend on a chronic inflammation of the capsule of the liver, or, as it is termed, perihepatitis, partly owing to the size of the organ and partly because, with the urine healthy, there is little likelihood of the presence of this condition. Thrombosis of the portal vein from adhesive portal phlebitis is said to have caused ascites, but this would not affect the size of the liver much. It appears to me that portal obstruction from some form of cirrhosis is the most likely assumption. Alcoholic cirrhosis occurs sometimes in children, their fibrous tissues involved in this condition being easily excited by very slight stimuli to irritative proliferation. The child, however, says she has never tasted beer, wine, or spirits. In the early stage of alcoholic cirrhosis there is often, if not always, some degree of enlargement of the liver, which subsequently may become smaller than normal; but whether the liver in a case of cirrhosis is atrophic or hypertrophic is a matter of accident, the most typical atrophic form—the alcoholic one—being sometimes large, and the most typical hypertrophic—the biliary—being sometimes small. When considerable enlargement is present, the organ is almost always loaded with fat. Jaundice is more frequently absent than present in cases of cirrhosis of the liver. Some cases of cirrhosis in children follow, and appear to be a sequel of scarlet fever, but there is no history of scarlet fever in this case. The dyspeptic symptoms, with occasional vomiting, which occurred fifteen months ago were not preceded by any other illness.

Diffuse syphilitic cirrhosis is met with in children who are the subjects of inherited syphilis. The spleen is said to be always enlarged as well. The liver is large and smooth, with an opaque capsule, tough, and on section is seen to have

undergone complete fibroid transformation in some parts. Microscopically, the lesion is seen to be diffuse, and not restricted to any anatomical region. There is, however, no history of inherited syphilis in this patient, and the other signs of this cachexia, namely, the depressed nasal bones, interstitial corneitis, and half-moon central upper incisors are absent. Tuberculous cirrhosis has also to be considered. The condition is generally admitted by German pathologists. It is said to occur in tuberculous patients. Dr. Saundby says: the liver is enlarged, smooth, and of normal consistence, but, on section; a trabecular network is seen to surround the lobules, and under the microscope this is seen to be of great extent, and accompanied by a most remarkable development of biliary canaliculi. Dr. Saundby also describes a form of cirrhosis which he calls rachitic, which occurs in rickety children. An important feature in its etiology being chronic gastro-intestinal catarrh, occurring in early childhood. The symptoms are those of intermittent gastro-intestinal catarrh. The child's appearance is cachectic, as in our case, and the disease has a strong superficial resemblance to one of congenital syphilis; but there is no history of syphilis, and there are no specific signs. The liver is enlarged, smooth, and of normal colour, or pale from fatty infiltration. On section it is tough, and traversed by trabeculae of fibrous tissue, which surround single acini, and are accompanied by a considerable new formation of biliary canaliculi. The spleen is said to be also enlarged. The diagnosis of the case I have brought before you is difficult, if not impossible, but I am inclined to favour some form of cirrhosis. The treatment adopted has been, first of all, to attend to early dyspeptic symptoms. During the last nine months the child has taken cod liver oil twice a day, with the addition of ten drops of carbonate of creasote to each dose. For the same length of time she has had daily applications of linimentum hydragryi spread upon flannel, large enough to cover the abdomen. This remedy has also been sometimes rubbed in as well. There has never been the slightest symptom of pytalism, although the patient has, upon two or three occasions, taken calomel internally as a diuretic. It is astonishing how difficult it is to salivate a child. Of course, one never wishes this to occur. The

diet allowed has been a liberal one, which has included cream and milk. Fortunately, the appetite has usually been good, with a clean tongue and regular action of the bowels. Since the onset of ascites, aperient medicines which produce watery excavations, have been given, and diuretic mixtures as well. One of the best of these is considered to be copaiba resin, but it did not do much here, although, as I have told you before, the kidneys are not diseased. Neither did the pill of digitalis, mercury and squill, increase the flow of urine, even when given soon after tapping. Diaphoretics have also been prescribed.

I must confess that I think that this plan of treatment often fails—I mean relying upon drugs which act upon the emunctories—it is generally by a more tonic mode that good results are obtained. It has been suggested that all forms of ascites should be treated by an entirely fluid diet—four or five quarts of milk daily (you must not forget that milk is a considerable diuretic), combined with frequent tapping and the administration of iodide of potassium, and of course proscribing all alcohol. I have recently given this child three grains (increased to five grains) of urea three times a day as a diuretic, but with no satisfactory result. The quantity of urine passed in twenty-four hours varies from six to fifteen ounces only. The profession is much in favour of early and frequent tapping for ascites, no matter what it arises from. The *rationale* of this is to encourage the establishment of a collateral venous circulation between the portal and systemic vessels. There are at least two disadvantages to this, one being that the products of digestion pass directly into the general venous system without being altered by the liver, and the other is that the existence of large and thin-walled varicose veins in the mucous membrane of the œsophagus are very liable to rupture, leading to profuse hæmorrhage into the œsophagus or stomach. It has been said that in cases of cirrhosis those patients who have abdominal dropsy usually escape hæmatemesis, and those who have hæmatemesis are generally free from dropsy of the abdomen. The development of a collateral circulation around the lower end of the œsophagus, by which portal vein blood can reach the heart without passing through the liver, is now well understood. This relieves the portal engorgement which is likely to arise in cirrhosis of the liver.

There is a reversal of the normal flow of venous blood, which is discharged into the general venous system by short diaphragmatic veins; by the œsophageal branches of the intercostal veins; and in the upper part of the œsophagus by branches of the bronchial and the inferior thyroid veins. This plexus is outside the muscular coat of the œsophagus. There is another plexus in the mucosa and submucosa of the cardiac orifice and œsophagus, branches from which pierce the muscular coat, and so communicate with the systemic veins.

The enlargement of the veins on the surface of the abdomen is due to an enlargement of the epigastric veins in connection with the opening up of the old umbilical vein, which often becomes as large as a goose quill. Through this the portal blood flows and finds a free passage into the vena cava and systemic circulation. As in the œsophageal flexus so here, injections may be easily made to pass from one system into the other. The interval between the tapplings has increased very much of late, and is accounted for by these collateral circulations.

The main question of treatment for us to consider is whether an operation is justifiable. Simply opening the abdomen and washing it out with antiseptic fluid has been known to do great good in cases of tubercular peritonitis. One case reported by Sir Spencer Wells made a perfect recovery after passing through an attack of acute peritonitis. But supposing the ascites in our case to be related with some form of cirrhosis, we can, I think, give the patient a fair chance of recovery by producing an accessory portal circulation. It has been noticed at post-mortem examinations that cases of cirrhosis, in which the portal circulation had been relieved through new anastomatic vessels developed in adhesions, binding the viscera to the parietes, had no ascites. Mr. Rutherford Morison reports that he has on two occasions tried to imitate this natural cure by opening, drying, and draining the abdomen, stitching the omentum to the abdominal wall, and strapping the abdomen firmly; thus keeping the sponged surfaces of liver, spleen, and intestines in contact with the parietes, and allowing adhesions to form. The first case failed, and the patient, more than a year after, had still to be tapped once a fortnight. His second case might, so far, be described as a

brilliant success, for the patient, worn out and emaciated to the last degree, and requiring to be relieved of from four to five gallons of fluid every few days, was now—two months after the healing of the drainage-tube opening—free from any swelling, and apparently in good health. I have mentioned these cases to Mr. Greig-Smith (the surgeon to the case), and he tells me that, although he has not done the operation, he thinks it would be most suitable; but he says better adhesions would be obtained by stripping the peritoneum, and so, when the child recovers from a slight attack of lung congestion, he intends operating with this modification. Up to the present there has been no attack of hæmatemesis; should it occur, the routine plan of giving ergot hypodermically should be avoided, as it does harm by driving the blood from the arterial into the venous system, besides increasing the heart's work. It has been suggested to adopt an exactly opposite line of treatment, and by means of nitrite of amyl, or nitroglycerine, or, perhaps, if it is desired to keep up the action of the drug for a longer time, erythrol nitrate, to rapidly draw a large amount of blood into the capillaries. The bursting of these distended veins may be assisted by some slight ulceration; and, whether this is so or not, one's object is to get the vein firmly plugged before anything is put into the stomach. Therefore it is best to feed these patients for at any rate a couple of days with rectal nutrient enemata, and see that the bowels act freely once in twenty-four hours.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London
Clinical Society, North-West London Hospital,
February 19th, 1896.

DR. MILSON in the Chair.

Post-hemiplegic Paralysis Agitans.

Dr. GEORGE JOHNSTON exhibited a man of about 65, the subject of post-hemiplegic paralysis agitans, who had been under his observation 18 months. He said the interest of the case did not lie so much in the patient's present condition as in the

mode of onset of the illness. About eight years ago he went to bed in perfect health, and awoke in the morning to find himself absolutely paralysed on the right side. During the following two or three days there was aphasia, and the arm and leg remained completely paralysed for at least two months. Then power commenced to return first in the leg, and with it tremor began in the limb. About a fortnight subsequently, tremor was evident in the right arm, and had persisted, and even increased up to the present time. Up to 18 months ago there was practically no shaking on the left side, so that this symptom remained confined to the right side for more than seven years. The question arose, and would have arisen more poignantly a year and a half ago, as to whether the case was really one of hemiplegic paralysis agitans, or post-hemiplegic hemichorea, because there were instances of the latter disease which very closely simulated the former. In such cases as the present, where post-mortem examination had been made, it had been found that the lesion was in the basal ganglion, in the neighbourhood of, and involving the optic thalamus and posterior part of the internal capsule. Those cases had almost invariably been accompanied by hemianæsthesia of the paralysed side, as would be expected from the situation of the lesion. But in the present patient no hemianæsthesia was apparent, though he himself said there was a certain loss of sensation on the right side at the time of the onset of his illness. Again, these cases of post-hemiplegic chorea have almost always remained confined to the paralysed side, and the other symptoms of paralysis agitans have been found wanting.

In support of his view Dr. Johnston called attention to the stooping posture when walking, the bent position of the arms, the working of the fingers upon one another, and the fixed stony expression of the face from the lips upward. In addition, the patient had both propulsion and retropulsion, the spontaneous propulsion being a source of especial annoyance. Lateropulsion was also noticeable, *i.e.*, the man would take one or two unnecessary steps to the left, and in going out of a door would sometimes encounter the door-post. Besides those symptoms he had a great sense of heat, and at one time restlessness was very well marked. A point of interest was the

relationship which the cerebral lesion which must have caused the hemiplegia bore to the paralysis agitans.

Several other cases of paralysis agitans were on record which had followed upon hemiplegia, and the question was whether the site of the lesion had anything to do with it. Erb thought it had, and reasoned by analogy. The lesion was found in the posterior part of the internal capsule, and in the neighbourhood of the basal ganglia, involving the optic thalamus. Accepting this, it would be difficult to account for the spread of the symptoms to the other side of the body. Erb tried to get out of that difficulty by presuming that, in his case, the lesion was in the pons (not in the internal capsule), where, of course, the motor tracts of the opposite sides were in close relation to each other. But it was difficult to explain the fact—present in both Erb's case and in the present one—that after the lapse of seven years, the tremors should have spread to the opposite side. He (Dr. Johnston) thought it more likely that the lesion was the determining cause rather than the actual cause; that the man was already predisposed to paralysis agitans, which, as far as was known, was a functional disorder, and that the shock of the hæmorrhage acted as other shocks did in predisposing to disease. Another interesting point was the practically entire absence of the knee-jerks, both right and left, and this had been noticed during the whole 18 months the man had been under Dr. Johnston's care.

Dr. CAGNEY agreed that the case in its present form was one of typical paralysis agitans, and it was additionally interesting regarding its relationship with or apparent origin in hemiplegia. He did not recommend too close an association of the two conditions. As to Dr. Johnston's speculative reasoning concerning the later condition of paralysis agitans being possibly caused by changes in the cerebrum due to the hæmorrhage eight years ago, he did not think our knowledge of the changes which follow gross injury in the cerebral centres justified more than speculation of a very cautious kind, though it was a matter of great importance. Both the diseases which had been mentioned were forms of degeneration, and the hæmorrhage might be supposed to be vascular degeneration, no doubt due to a weakening of the vessel walls or by strain behind them, rather

than to changes in the nervous system. But it was known that people undergoing cerebral hæmorrhage were usually the subjects of degeneration of extensive character, involving the whole of the nervous system.

Such people sometimes got other forms of peripheral affection, and he believed that paralysis agitans involved a peripheral, as well as central, degeneration. From the comparatively few cases of paralysis agitans following hemiplegia, he thought they might be regarded as illustrating a coincidence depending upon the degenerated tendency. But such cases of coincidence required watching, for by this means a great deal might be learnt. The absence of the knee-jerks in this case was, perhaps, connected with the central lesion. He remembered having had under close observation an old lady, who had had an attack of hemiplegia, and who was also a subject of glycosuria. After eighteen months of hemiplegia, there was no post-hemiplegic contracture, a fact remarked upon by several eminent physicians, including the late Prof. Charcot and Sir Wm. Broadbent. He thought that absence was explained by the electrical reaction, which showed a degree of reaction of degeneration on the side which had been paralysed in consequence of the hemiplegia. He reported the case, as bearing out what he thought, at the time, to be his own theory, that the muscles and other peripheral structures were dependent, primarily, on the motor ganglia of the spinal cord, but to a high degree also, and in a secondary manner, upon the efficiency of their communication with the cortical centres. He had not heard of other cases since, but in reading later he found there was ample ground for the view that profound modification of the cortical centres will greatly alter the nutrition of the muscles, and, he supposed, of the bones and secretory organs also, whose dependence upon the spinal centres we knew less about. Therefore, the absence of the knee-jerk should be taken into account in any explanation attempted.

Dr. HARRY CAMPBELL asked whether the whole of the symptoms of paralysis agitans appeared after the hemiplegia, or only the more obtrusive symptom of movement. A person might have the disease coming on for many years, but the agitative movement might not be developed until the end of the disease.

Dr. MACEVOY said it had not been stated whether there was any peculiarity of speech in the patient, a point he would like information upon.

Dr. JOHNSTON, in reply, said the voice was monotonous, inclined to be "piping." Dr. Cagney, apparently, took practically the same view of the case as he himself, viz., that there was a predisposition to the state of paralysis agitans, and that the cerebral hæmorrhage, which had probably occurred in this case, acted more as a determining cause, but that it was also due to degeneration, in the way that lack of nutrition of the cortical centres was also an evidence or a cause of degeneration. He could not quite regard it as a coincidence, but was more inclined to look upon it as a cause. He did not believe that paralysis agitans would have occurred at that time had the cerebral hæmorrhage not happened.

Replying to Dr. Campbell, as the onset of the illness was eight years ago, he could not be certain that the other definite symptoms of paralysis agitans became immediately present; still, although the tremor might be absent all through in paralysis agitans, some amount of tremor was present as a general rule. The fact that other cases had been exactly similar in their course, the shaking commencing with returning power, was of importance.

Suppuration in Tympanic Cavity.

Mr. MAYO COLLIER showed a boy, æt. about 7, with suppuration of the tympanic cavity, of fourteen days' duration. An abscess of the mastoid process was evident, but he did not wish to intimate that there was acute retention of pus causing pressure. There was a total absence of cerebral symptoms, the temperature was normal, and there was no pain. In many cases an affection of the periosteum on the outer side of the mastoid process was met with, but in this case he did not expect to find dead bone when opening the abscess. If the abscess was opened and disinfected he thought the boy would get well. In some cases in which pain was present, necrosis in the mastoid cavity was found, and in those it would be wise to make a free opening. If the present abscess were opened at once, drained completely, and ventilated through the auditory canal, it would probably heal straight away.

Mr. JACKSON CLARKE showed a boy on whom the operation just described by Mr. Mayo Collier had been recently performed, the only evidence of the operation being a linear scar. The ear, which, before the operation, was pushed from its place, had now returned to its normal position. He exhibited several bones showing the results of operations, and the relation of the tympanic cavity to its surrounding parts. In one case, which was cured by opening the mastoid antrum, the only drawback was that, about thirty hours after operating, although there was no paralysis previously, facial palsy of slight degree was noticed, which gradually increased until complete, and then, in a few days, passed as completely away. It was probably due to temporary oedema of the nerve; and, possibly, in scraping out the tympanic cavity with the curette, a little of the nerve was exposed, where the bone had been eaten away by granulation tissue.

A Double Cryptorchid.

Mr. MAYO COLLIER showed a boy, whose history was as follows:—The mother noticed a swelling in the left inguinal region some twelve months ago, and applied to another London hospital for advice and relief. He did not know what the advice was, but the relief consisted of supplying a truss, the boy being considered to have inguinal hernia. No trace of such a condition could be found; there was no opening in the external abdominal ring, nor any obliterated sac. But he found a testicle situated in front of and a little below the external abdominal ring. The truss had been worn for nearly 12 months, and recently a swelling appeared in the other groin, and application was made at the North-West London Hospital for a truss for the other side, when he found a testicle on that side also. It simulated inguinal hernia in some respects.

Aphonia and Chronic Laryngeal Catarrh.

This case of aphonia in a woman of about 35 was also shown by Mr. Mayo Collier, who said that he and the resident medical officers of the hospital were justly proud of the cure which had been effected. The symptoms were of seven or eight months' duration. The persistent dryness of the throat, the cough, the aphonia, and loss of

sleep had so wrought on the patient that her health seriously deteriorated. She had suffered more or less all her life from hoarseness, cough, and throat trouble, with consequent indifferent health, particularly in the winter. When she presented herself lately, his note was: chronic laryngeal catarrh, hoarseness, loss of voice, chronic nasal obstruction on both sides. On examination, he found chronic hypertrophic laryngeal catarrh, the large velvety appearance of the arytenoid cartilages, running all along the arytenoid folds. In the pharynx the glands were enlarged, and venous radicles coursing over the parts. The post-nasal region was extremely irritable, and proper examination was practically impossible, because a quantity of mucus came pouring down from the nasal cavity. Examination of the nose showed absolute blockage on the right side, due to deflection of the septum, and the anterior half of the bony septum pressing upon the terminal bone, causing blockage. On the left side was a turbinal varix as large as a pigeon's egg. He had treated the case with an antiseptic wash, which had been used twice a day for some months. In addition to that, he had treated the post-nasal space by (?) electrolysis, and benzoin had been ordered for her cough. Moreover, Mr. Horsford and Dr. Lewis had undertaken to galvanise her every day. He (Mr. Collier), however, was certain no good would be done until the physiological function of the nose was restored. He therefore cauterised the right side. In three days afterwards the patient began to get a slight amount of voice (the gradual return disproving any idea of hysterical aphonia). Further treatment was then stopped, and by the answers the patient gave to questions, the members could judge how complete had been the cure.

Mr. JACKSON CLARKE expressed his personal obligations to Mr. Collier for his interesting narration of the case, and spoke of the practice, in some parts of London, of removing the inferior turbinated bone for varix. That, he thought, was a rather severe mode of dealing with a condition which was not due to any permanent structural change. He would like to ask how the electrodes could be applied where the mischief was located at the posterior extremity of the inferior turbinated bone.

Mr. MAYO COLLIER, in reply, said he strongly

disapproved of removing a structure which filled a definite purpose. He held that the object of the erectile tissue of the turbinated bones was to warm and moisten and filter the air, and the removal of the turbinated for varix left the patient in a worse condition than before. Mr. Collier replied to Mr. Jackson Clarke by describing fully the *technique* of the operation. He had never yet removed a turbinated bone for varix. He once heard a surgeon state that he had removed 500 turbinated bones, and he took the opportunity of remonstrating with him for what he conceived to be unjustifiable interference.

Unusual Syphilitic Eruption.

Dr. SIBLEY showed a woman, æt. about 35, presenting an extensive syphilitic eruption between the shoulders. It was more extensive than usual, because she had been laid up with a cold and had not taken iodide of potassium during that time. She had been under treatment more or less continuously for twelve years. The eruption had gradually spread at the periphery, leaving a scarring behind, and in many respects it simulated lupus erythematosus. It had been present in the hair, scalp and face, but always subsided under iodide treatment.

Dr. HARRY CAMPBELL said he remembered the patient being under his treatment five or six years ago, and during that time she became pregnant. He plied her assiduously with antisymphilitic drugs, and the child was born quite healthy. He would like to hear Dr. Sibley's views as to the period at which a syphilitic mother ceased to infect her offspring.

Dr. SIBLEY said the woman had borne four children before the onset of syphilis, one of whom had died rather early in life from convulsions. She had had two children since; neither had signs of the disease, though the one had some scars on the leg. He held no opinion which would enable him to reply to Dr. Campbell's question.

A Case of Marked Presystolic Murmur.

Dr. HARRY CAMPBELL showed a young woman, æt. 24, with a loud presystolic murmur and thrill. There was very definite mitral constriction, and the pulse tension was high. Sir William Broadbent had pointed out that in cases of presystolic

murmur the tension was very high, but he (Dr. Campbell) had not always found it so. This case, however, was an illustration of Sir William Broadbent's statement.

A Case of Paralytic Tremor.

Dr. CAGNEY showed a man of about 60 years of age, the subject of tremor. His hands, knees, and ankles were swollen, and a good deal crippled by rheumatoid arthritis, while there was some suggestion of true gout. He was a clerk, and admits to having indulged too freely in alcohol at one time. He denied syphilis, but he (Dr. Cagney) was not clear that he had not had it. The palsy of the right hand, though of slow growth, was sufficient to prevent him following his occupation; and there was slight strabismus and lateral deviation of the eye, with impairment of the left rectus. There was some asymmetry of the face also, and the right half was usually tremulous when the muscles were in use. There was no nystagmus or pupillary disorder. The knee-jerks were exaggerated, there was no ankle-clonus, and the patient was uncertain of his equilibrium, having a tendency to fall or move backwards—what was called retropulsion. The countenance was fixed in a stony stare, and the gait was that of paralysis agitans.

There were also the iliac pains, restlessness and hot flushes belonging to that complaint. The tremors, however, did not correspond in character or rhythm. They were of hemiplegic distribution. This was usually so in the early stages of paralysis agitans, but they were increased and not diminished by purposive action. At the onset his diagnosis had been probable disseminated sclerosis, but there was a progressive and growing resemblance to paralysis agitans. He had produced the patient as a companion picture to Dr. Johnston's, and, as in that case, there was possibly a combination of lesions. It was notable that, whereas formerly the patient had suffered much pain from rheumatism, this had ceased with the incidence of the present malady.

Dr. CAMPBELL thought the case was a typical one of paralysis agitans. Charcot had described a case of a woman in whom the disease appeared suddenly on seeing her husband's horse return riderless, and in another the cause was the sudden

attempt to save her child from fire. He agreed that the case, in its early stage, showed signs of disseminated sclerosis.

REVIEWS.

Diseases of the Joints and Spine. By HOWARD MARSH. (Cassell & Co.)

We cannot help feeling some little disappointment in this book; it is excellently written, most clearly expressed, and certainly ought to give great satisfaction, and yet somehow it seems incomplete: probably the fault lies with the subject and not with the writer.

There are many excellent chapters: Chapter XI, on syphilitic diseases of joints is one of the best, and is written entirely from experience and not theory; the chapter on bone-setting is also instructive, we had almost said amusing. It is the chapters on gout, rheumatism, and osteo-arthritis that we think are weakest, possibly because the book is written by a surgeon. We have, however, no hesitation in strongly recommending the work as of great practical assistance to any one in dealing with a case of joint disease; Mr. Marsh is evidently as conservative of limbs as the disease will allow.

Heart Inflammation in Children. By O. STURGES, M.D., F.R.C.P.

This little book is a reprint of the Lumleian Lectures for 1894. Like everything that its lamented author wrote, it is characterised by precision and completeness, and he traces from Lænnec the history of the recognition of cardiac disease in children. It is not all historical, for the clinical picture of a child's attack of heart mischief is painted with a master hand, and the most experienced will feel the keenest delight in this reproduction. To those interested more especially in heart disease the book is a necessity, but to the general practitioner we fear it will be only a cheap luxury, but one we should like to see enjoyed by all, for its perusal will be a mental treat, not unaccompanied by a real gain in practical knowledge.

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 11, 1896.

CLINICAL LECTURE

ON

TWO CASES OF THORACIC ANEURYSM.

By W. HALE WHITE, M.D., F.R.C.P.

Delivered at Guy's Hospital, Feb. 1, 1896.

GENTLEMEN,—The two men you see before you are both suffering from a thoracic aneurysm, and to-day we will go over the chief points of interest in this disease.

Case 1.—A stoker, æt. 30, who has had very hard work, who has drunk freely, and who has had ague frequently, was admitted for severe pain in the left chest and difficulty of breathing. About seven months ago he first noticed pain in the upper part of his chest on the left side, and a month later he was seized with an attack of coughing, and brought up some blood. Since then his voice has been weak, he has been troubled much with cough and shortness of breath, he has often brought up blood, and the paroxysms of pain starting in the upper part of the left chest and radiating into the left side of the neck and to the left scapula, and down both arms, especially the left, have been agonizing and so constant that he cannot sleep, and now he is in continual pain, which is only varied by the frequent onset of extremely severe paroxysms. He complains of difficulty of swallowing.

If, now, you look at him you will see that his very expression shows the constancy and severity of the pain. He spends most of the day sitting up resting his head on his hands, and coughing frequently.

Case 2.—This man is a waterside labourer, æt. 40, who came in for pain in the back. He has had syphilis, has drunk frequently, and worked hard. About four months ago he began to complain of cough, and about the same time he had a gnawing pain in the back between the left scapula and the spine. Not long after this he began to suffer from shortness of breath and cough, and he soon had to give up his work.

VOL. VII. No. 20.

If we now examine the chest in these two cases, you will see that in both there is an area of dulness in such a position that it might be due to an aneurysm, and whenever that is the case you must carefully seek for any other signs of a thoracic aneurysm, and as you do so you must consider whether they are due to an aneurysm or an intra-thoracic growth. We will now do this, and the symptoms may be roughly divided into those due to the presence of a vascular tumour, direct pressure signs, and reflex pressure signs.

SYMPTOMS OF THE PRESENCE OF A VASCULAR TUMOUR.

1. *Dulness.* As already mentioned, you must carefully consider whether this is in such a position that it could be produced by an aneurysm. In our first case it extends upwards, and to the left from the third part of the arch, and in the second it is to the left of the spine, just above the angle of the scapula, and when a thoracic aneurysm occurs on the descending thoracic aorta, this is the commonest seat for it.

2. *Tumour.* In our first case there is slight prominence of the upper part of the left chest, and in the second you can see a large prominence in the back, the size of a very large orange. If the aneurysm is deep-seated, and is not growing towards the surface, a tumour is not seen during life.

3. *Pulsation.* You must examine this most carefully, for mistakes are very easy. If it is due to an aneurysm, the pulsation is expansile, as you can see beautifully in our second case. Growths so vascular as to cause expansile pulsation are so excessively rare in the chest in comparison with aneurysm that expansile pulsation is of the utmost importance in pointing to an aneurysm. On the other hand, if the pulsation is simply transmitted, and therefore non-expansile, then we are dealing with some extraneous tumour pressing on a vessel, and not an aneurysm. The only fallacy here is one that I was able to show you a year ago, namely, that when the aneurysm is completely full of blood clot, it becomes really an extraneous tumour

on the vessel, and the pulsation is transmitted and not expansile, but this is very rare.

4. *Systolic Murmur.* There is nearly always a systolic murmur, usually soft and blowing, over an aneurysm, and it is a very important sign. Our first patient has one, but our second case has not, probably because the orifice of his aneurysm is very large. You must always try to make out from other signs that it is not due to pressure of a tumour on a vessel, nor to cardiac disease.

5. *Diastolic Murmur.* It is excessively rare for this to be produced in an aneurysm, but it may exist. Whenever you hear a diastolic murmur it is nearly always due to the fact that the patient has aortic valvular disease as well as an aneurysm, and as the pulse is somewhat aortic, and the heart is much hypertrophied, this is the most likely cause of the diastolic murmur heard in our first case. In quite exceptional cases an aortic aneurysm ruptures into the pulmonary artery, and then if the patient survive there is sometimes a diastolic murmur. The fact that our patient got suddenly much worse while lifting a weight suggests this cause for his murmur to us. Our second patient has not got a diastolic murmur.

6. *Thrill.* Neither of our patients present this symptom. A thrill is hardly ever produced in a sacculated aneurysm; and if you feel one which is, from its position, not caused by disease of the heart, it is almost certainly due to either a dilated arch of the aorta, pressure of a tumour on a vessel, or some communication between the pulmonary artery and the aorta.

7. *Accentuated Aortic Second Sound.* In neither of our patients can you hear this, but when the aneurysm is near the aortic valves you often may, for the mass of blood thrown into the aneurysm at each systole drops back suddenly on the aortic valves, and so brings them together very sharply.

8. *Early Diastolic Shock.* This, too, is absent in our cases, but it may be noticed in cases in which the aneurysm is near the aortic valves if you place your ear on a wooden stethoscope which is on the aneurysm. It is produced in this manner. The blood in the aorta which has fallen on the aortic valves closes them, and the instant they are closed a recoil wave starts back from them; this meets the blood pouring out of the aneurysm—for if this has a narrow orifice it takes some time

to empty—and hence this recoil wave temporarily checking the emptying of the aneurysm, produces a shock felt by the ear in the early part of the diastole.

9. *Shock of Impulse.* To appreciate this you must also use a wooden stethoscope. It is merely the transmission to your ear of the expansion produced by the blood entering the aneurysm. It is noticeable in both our cases, but is of little value, for it may be felt over a tumour which presses on a vessel, but the seventh and eighth signs are almost conclusive proof of the presence of an aneurysm.

10. *Systolic Murmur over the Trachea.* If the aneurysm is in contact with the trachea or a large bronchus these structures conduct the aneurysmal murmur very well. On placing your stethoscope over the trachea you can often hear the murmur: it is very audible in our first patient.

11. *Systolic Murmur Audible in the Mouth.* When the murmur is loud and the trachea conducts it up very well, if the patient puts the chest-piece of a binaural stethoscope into his mouth and shuts it around it you can sometimes hear the murmur. In our first patient it is very loud.

12. *Tracheal Tugging.* When the aneurysm is so placed that each expansion of it presses down one of the bronchi, the whole trachea is pulled down, so that if the patient sits up with his head thrown back, and you stand behind him and place the forefinger of each hand under his cricoid cartilage and pull the trachea up, at each systole you can feel it tugged down from you. It is so marked in our first patient that I think you can see my fingers pulled down. These last three signs are very strong evidence of aneurysm.

PRESSURE SIGNS.

1. *Pain.* This is of two sorts—local, which is caused when the aneurysm, coming to the chest wall, presses directly on the sensory cutaneous nerves, and radiating, which is due to pressure on the nerves supplying the diseased aorta. The pain radiates over those cutaneous areas which are supplied from the same segment of the cord as the affected part of the aorta. Both patients have the local pain very well marked. The second case has not much radiating pain, but in the first patient it is agonizing. When radiated pain is very severe

it spreads into areas of skin which are supplied by segments of the cord contiguous to those in connection with the diseased part of the arch of the aorta, and so in the first patient, his fearful pain spreads not only over the front of both sides of the chest, and down both arms, especially the left, but also over the whole of the left side of the neck.

2. *Cutaneous Tenderness.* You know that when any viscus is diseased you may often find that the cutaneous area supplied by the same segment of the cord as the diseased viscus is tender. Thus, in aortic disease you get a definite tender area down the inner side of the left arm. These tender areas were first fully worked out by Dr. Head. In our second patient they are absent, and in our first, owing to the severity of his pain, the tender area is so wide that, like the reflected pain, it has lost much of its anatomical significance, for the whole area over which he feels pain is tender when touched with the head of a pin.

3. *Anæsthesia.* At first sight you might think that when, as a result of pressure, one of the intercostal nerves was destroyed, there would be much cutaneous anæsthesia; but this is not so, for you will remember that the areas of distribution of the cutaneous sensory nerves overlap, so that there is no piece of skin which is not supplied by two or three nerves. An aneurysm rarely presses on enough nerves to cause cutaneous anæsthesia, but in our second case enough intercostal nerves are destroyed for him to have a more or less irregular band of anæsthesia an inch or two wide round his chest. Please remember that this sign is very rare. All these first three pressure signs may be present in growth.

4. *Pressure on the Left Recurrent Laryngeal Nerve.* This, as it winds round the arch of the aorta, is very frequently pressed upon by an aneurysm. The abductor and adductor fibres run in separate bundles in the nerve, and are so placed that the first effect of pressure on the nerve is to paralyse the abductor, with the result that the left cord is adducted to the middle line, and does not move during inspiration. This produces no effect on the voice or speech. Later, both sets of fibres are paralysed, and the cord, as in our first patient, lies in the cadaveric position, midway between abduction and adduction, and the healthy cord moves over beyond the middle line in phonation. The obvious effect of this paralysis is that the

voice is low pitched and hoarse, and cough is difficult. You will notice these effects in our patient. It may help your memory to observe that the effect of stimulating the cortex is the same as the first effect of pressure on the recurrent nerve, namely, the cord is adducted. This is because the centre for adduction is more excitable than that for abduction, and very close to it. In some animals they can be separately excited, and in those in which they cannot, if the adductor fibres in the recurrent nerve are cut, stimulation of the cortex produces abduction.

5. *Sympathetic.* The fibres leaving the spinal cord by the first left dorsal nerve, and going up the cervical sympathetic trunk are often pressed upon by a thoracic aneurysm. If, as in our first case, the pressure is sufficient to paralyse the nerve, the symptoms produced are :—

(a) *Contraction of the Pupil.* This is by far the most constant symptom, but you must remember that even during health the two pupils may vary considerably in size. The contracted pupil will react to light, accommodation, atrophine and eserine; the contraction is permanent so long as the pressure remains.

(b) *Narrowing of the Palpebral Aperture.* This is very rare; our patient does not show it. The narrowing is due to the contraction of the smooth muscular fibres which exist in the upper and lower lids, the lower lid ascending a trifle and the upper lid descending, although it can still be raised voluntarily by contraction of the levator palpebræ.

(c) *Retraction of the Eyeball.* This, though seen experimentally in animals, is excessively rare in man, and even when present is very slight. It is probably due to the paralysis of the fibres of Müller's muscle which lines part of the orbit, especially the aperture between it and the sphenomaxillary fossa.

(d) *Slight Internal Strabismus.* This I have seen once, although our present patient does not show it. It is due to the fact that the external rectus gets a slight supply from the sympathetic.

(e) *Vascular Dilatation.* This, too, our patient does not show; it is very rare in

man, but I have seen it. When present it is much more limited in man than in animals, for it is confined to the side of the face and the ear. The retinal and conjunctival vessels are never affected in man.

(f) *Rise of Temperature.* This is a result of the last, and is therefore only noticed when the vessels are dilated.

(g) *Diminution of the Secretion of Sweat.*

(h) *Diminution of Salivary Secretion.*

(i) *Diminution of Lacrymal Secretion.* All these three unilateral effects have been observed in animals, but they are very rarely met with in man. Our patient considers that all three secretions are very slightly increased on his paralysed side, but probably he is in error.

(k) *Nutrition.* In young animals there is a slight want of growth on the side on which the nerve is paralysed, but, as far as I know, this has never been observed in man. It is clear that all these sympathetic symptoms might be produced by a tumour as well as an aneurysm.

6. *Phrenic Nerves.* It is excessively rare for either an aneurysm or a growth to cause any symptoms due to pressure on these nerves.

7. *The Spinal Cord* may be compressed by an aneurysm of the thoracic aorta, and then the patient will suffer from the consequent sensory and motor symptoms. They could hardly be present in our first case, but it is rather surprising that we do not see them in the second. Growth may produce the same effects.

8. *Unequal Radial Pulses.* You will often see great stress laid upon this as a sign of aneurysm, but its importance is much exaggerated. I have often pointed out to you that healthy people may have unequal pulses, and, as in our first case, they may be quite equal when there is obviously an aneurysm present. When an aneurysm makes the pulses unequal it does so either by direct pressure on the innominate artery or the left subclavian, or by altering the shape of the orifice of one of these vessels. It is a rare symptom of growth.

9. *Enlarged Veins* rarely result from pressure of an aneurysm, because it is difficult to obstruct the circulation through veins by slowly increasing pressure from without. Neither of our patients show enlargement of the veins. On the other

hand, dilated veins are common in growth, for malignant disease appears to have a special predilection for growing into veins and obstructing their calibre, so that whenever the superficial veins are very prominent the great probability is that you are dealing with a case of growth.

10. *Œdema.* This goes with the venous distension, so it is rare in aneurysm. It is usually the arm which shows the œdema.

11. *Thoracic Duct.* It is strange that, however much the thoracic duct is pressed upon by either an aneurysm or growth, no symptoms appear to result therefrom.

12. *Trachea.* Pressure upon this causes stridor and dyspnoea, and in rare cases a bulging may be seen on laryngoscopic examination.

13. *Bronchus.* Pressure upon this leads to dyspnoea, unilateral stridor, pulmonary collapse, and deficient entry of air into one lung. Often with an aneurysm there is bloody expectoration. This may be due to congestion of the bronchial mucous membrane or lung, or to the fact that blood oozes through the laminated clot of the aneurysm, at a spot where this has eaten its way through the wall of the bronchus. This expectoration of blood may go on for years before the patient dies. Our first patient has much dyspnoea and bloody expectoration. Growth will be just as likely as aneurysm to cause these symptoms, due to pressure on the trachea or bronchus.

14. *Vessels and Nerves at the Root of the Lung.* Either a growth or an aneurysm may lead to bronchitis, hæmorrhage into the lung, and not infrequently gangrene of it.

15. *Œsophagus.* Pressure upon this by either growth or aneurysm causes dysphagia, and consequent wasting.

16. *Lungs.* Either form of pressure on the lungs lead to displacement of parts of them, and the displaced portions become compressed and solid, and round these compressed parts there is much bronchitis. There must be considerable compression in our first case, because there is complete dulness in front, above the third rib, and behind, above the upper border of the scapula.

REFLEX SIGNS.

1. *Pueroxysmal Dyspnoea.* This is especially

marked in aneurysm, and our first patient gets very severe attacks of dyspnoea.

2. *Brassy Cough.* This is a very important sign of aneurysm, and almost diagnostic, but not quite so, for two years ago we had in the clinical wards a man with a typical brassy cough who had a mediastinal growth.

Often a patient with an aneurysm presents neither dulness nor a tumour, but then you will be guided in your diagnosis by the other signs we have mentioned.

RUPTURE.

A thoracic aneurysm may rupture into—

- | | | |
|-------------------------------|--------------|------------|
| 1. The trachea or a bronchus. | } The | commonest. |
| 2. The pleural cavity. | | |
| 3. The œsophagus. | } Rare. | |
| 4. The pericardium. | | |
| 5. Externally. | | |
| 6. The pulmonary artery. | } Very rare. | |
| 7. The superior vena cava. | | |

Rupture at one of the first five seats is immediately fatal. Cases which rupture into the pulmonary artery may be divided into three groups, viz., those which die immediately, those which die very shortly, and those which live some time. About a quarter of the cases fall in the first group, a quarter to the second, and a half in the third. This last is the most interesting. The patient tells you that he was suddenly taken much more short of breath than usual, and on listening to him you usually hear a long murmur, prolonged through both systole and diastole, and loudest at the left of the sternum, at the level of the third rib, where you feel a thrill. In a few cases only a systolic murmur can be heard, and in a few others there is heard over the aneurysm a to-and-fro murmur exactly resembling in character that usually noticed in aortic disease, but whatever murmurs you hear you nearly always feel a thrill. You will notice that it is possible that the to-and-fro murmur heard in our first case may be due to a rupture into the pulmonary artery, but I have already given you reasons against this view, and further, this patient has a thrill.

Rupture into the superior vena cava is excessively rare and is often fatal at once; but if the patient survive, you have a long swishing murmur on the right of the sternum, and you notice that

the lividity is confined to the head, neck, arms, and upper part of the trunk.

ÆTIOLOGY.

Often no cause can be assigned, and some persons are occasionally seen who seem to be especially liable to aneurysms in any part of their body, but aneurysms of the thoracic aorta are nearly always due to either syphilis or strain, or both, and some authorities consider that excessive indulgence in alcohol is sometimes responsible. When due to malignant endocarditis aneurysms are on small vessels—for instance, the last I saw were on the vertebral and the hepatic arteries—and, therefore, when dealing with an aneurysm of the thoracic aorta you need not consider malignant endocarditis.

DIAGNOSIS.

We have incidentally pointed out how to distinguish an aneurysm from a growth. You must also be on the outlook not to confound a considerable dilatation of the arch of the aorta with a sacculated aneurysm. Disease of the aortic valves is especially likely to give rise to a mistake, and when the aneurysm is on the intrapericardial part of the aorta a mistake is particularly easy. Curvature of the spine may lead to a wrong diagnosis in three ways. As a result of the curvature the second part of the arch may be displaced up and to the right; the dulness which is met with close to the curve may be mistaken for that of an aneurysm; and, lastly, the erosion of the vertebræ may lead to a curvature. At different times we have had examples of all these fallacies in the wards.

TREATMENT.

So few minutes are left that I can say but little about this; nor does it matter much, for, unfortunately, we know of no means of causing a clot to form in a thoracic aneurysm, and that is the only way a cure can be effected. In a few exceptional cases a natural cure takes place. Why these aneurysms should clot we are quite unable to say; they may do so under conditions apparently most unfavourable. Thus, not long ago, we had a case in which we were able to feel what we correctly, as it turned out, thought to be a cured aneurysm springing from the front of the

abdominal aorta. You would have thought a cure here impossible, considering that whenever the man lay down the tendency of the blood in the aneurysm would be to fall back into the aorta. On the other hand, you would think that if our second patient were kept on his back the conditions would be very favourable to clotting, but his aneurysm is growing rapidly, in spite of rest in bed; and the last one I saw in the same position grew very rapidly, although the patient was kept strictly on his back. The great discovery for someone to make is what is the factor that induces the clotting in the natural cures. One thing is quite certain, that the clot produced is quite different from the clot that can be produced outside the body; it is a hard laminated clot which cuts like cheese, and is not at all similar to the jelly-like clot which is seen in the laboratory.

Distal ligatures of the subclavian and carotid is never justifiable for a thoracic aneurysm, even if it seems to you to be confined to the innominate artery or the thoracic portion of either the left subclavian or left carotid, for in these cases the arch of the aorta is always implicated, the operation is very serious, and the vessels which it is proposed to ligature are usually bad. Nor is the introduction of foreign bodies, as wire, hair, or electric needles, to be recommended, for satisfactory clotting is not produced. I have seen the jagged ends of eroded ribs sticking into an aneurysm, forming most excellent foreign bodies, and yet no clotting took place. Some surgeons have passed needles into the sac and scratched the opposite side of it, but this is not often done.

Tuffnell's treatment has frequently been tried, but it is doubtful whether any benefit follows from it. I have never seen it do any permanent good. The patient may be better while he rests in bed, but that is all. More than once I have seen the aneurysm rupture a few days after the patient was allowed out of bed after months of rigid Tuffnell treatment. Iodide of potassium has been recommended, but this too appears to be of little benefit. It has been advised that patients should be fed largely on fat, but this also has failed whenever I have tried it. Calcium chloride is the last drug advised. It undoubtedly can, outside the body, increase the coagulability of the blood, but at present I have seen no good results follow its administration in cases of aneurysm. As the

inhalation of carbonic acid gas has been recommended, we are going to try it and give calcium chloride, but I am not very sanguine. In fact, all these modes of treatment are wrong in principle, for our object should be to produce a local clotting, while all these methods increase the clotting power of the blood generally. It is a melancholy conclusion, but I am afraid we can do nothing to cure a thoracic aneurysm.* It will be your duty to put this to the patient, and point out to him that if he continues his daily work he, in all reasonable probability, cannot live long; if he lies down and leads quite an idle life, he can live a little longer. I believe that most would, like Dr. Fagge, choose to die in harness.

Fortunately, you can do much to relieve the pain. Morphia is here your sheet anchor. Bleeding, too, often acts like a charm, but it has failed in our first patient; but he, like some others, finds his pain relieved by taking iodide of potassium. When the pain is paroxysmal, nitrate of amyl inhalations may benefit it.

A CLINICAL LECTURE

ON THE MODES OF

COMMENCEMENT OF PHTHISIS (PULMONARY TUBERCULOSIS).

By SIDNEY MARTIN, M.D., F.R.S., F.R.C.P.

Assistant-Physician to University College Hospital, and to the Hospital for Consumption, Brompton.

I PROPOSE to limit myself to-day to some of the clinical aspects of phthisis, which are of great importance in the recognition of the disease, and chiefly to a discussion of its modes of commencement. When a healthy animal is either inoculated or fed with tuberculous virus or with tubercle bacilli, the development of the lesion is very slow, and produces at first but few symptoms; indeed, there may be no symptoms until a very late stage of the disease. In experimental tuberculosis, therefore, the onset of the disease may aptly be

* The bad outlook in cases of aneurysm is shown by the fact that the second case died a month after the lecture was given.

described as nearly always insidious. The rapidity of the development of tuberculosis depends on three factors, which are always to be borne in mind when discussing its pathology or considering its clinical aspects. These three points are :

(1) The degree of virulence of the bacillus tuberculosis.

(2) The dose of the virus.

(3) The power of resistance of the individual (animal or man).

I do not wish to-day to discuss the pathological aspects of tuberculosis, except, perhaps, now and then to refer to the bearing of well-known facts on the clinical history of the disease. But I wish to dwell upon the aspects of tuberculosis, and especially of phthisis (pulmonary tuberculosis) when first observed by the physician. These aspects are very varied, and may be roughly classified as (1) whether the disease is first evidenced by a prominent symptom, or (2) is masked by groups of symptoms not referable to the lungs.

INSIDIOUS ORIGIN OF PHTHISIS.

The most common mode of commencement of phthisis is insidious, and the usual history obtainable from such patients is that, for a definite time—perhaps one month, more usually three or six months, or longer—there has existed a group of symptoms which we may fairly consider as indicative of the disease. Cough, for example, varying in degree; expectoration, at first slight and mucoid, and later becoming yellow and purulent; associated, in some cases, with hæmoptysis, but in nearly all cases with the occurrence of wasting and of night sweats. The characteristic night sweats of phthisis are not those which occur in the earlier part of the night, soon after the patient goes to bed, and which may be due to varying causes, such as over-eating, hot weather, etc., but are those which occur in the early morning, between one and two a.m. These night sweats are usually a sign of nocturnal fever, but they are not proportional to the fever, and frequently, after a few months of illness, they diminish or disappear. When a patient complaining of this combination of symptoms is examined, physical signs varying in degree are discovered. These will be considered later. As regards the early symptoms, individual patients present some variation. Although, if kept under

observation, they nearly all show at one time or another an evening rise of temperature; this may be very variable after the disease has lasted one or two months. Other cases present symptoms of excessive wasting, without much cough and only slight expectoration, and in these cases the presence of night sweats would indicate tuberculosis. In other cases the cough may be excessive; there is no expectoration, but wasting and night sweats are present. Both these classes of cases may present no hæmoptysis up to the time of the first examination, and the physical signs found in the chest may be doubtful, so that as regards symptoms one looks to the presence of night sweats in association with wasting and cough as indicative of phthisis. I think it important to insist on the characteristic symptoms of pulmonary tuberculosis, inasmuch as not infrequently these symptoms are unappreciated, and a careful examination of the chest, which, when made, would indicate the disease, has been omitted.

Turning to the physical signs which may be present in these early cases of phthisis, there is, first, a deficiency of movement at one apex; and a difference in the percussion note below the clavicles, that on the side in which there is deficient movement being shorter and higher-pitched than on the other side, or is actually dull, while on auscultation and comparing the breath sounds on the two sides, it is found that expiration is prolonged and respiration is divided, or that there is cog-wheeled inspiration on the diseased side. The signs of deficient movement at one apex indicate a deficient entrance of air into the upper part of the lung, and the difference in the percussion note, together with the prolongation of expiration, suggests slight consolidation. But in these early cases such physical signs are sometimes absent, are more frequently doubtful, and the chief signs to rely upon in the diagnosis of early consolidation are an increase in the vocal or tussive resonance at one apex or the presence of crepitations and rhonchi. As regards rhonchi, it may be said that these alone may be present, and you may hear a sibilus at the end of inspiration or coming out on cough, which, if present at the same place on two or three different examinations, would be indicative of the apex being tuberculous. The crepitations which are characteristic of consolidation are those which

occur during inspiration, but also those which are produced by the act of coughing, or are heard on the first inspiration following a cough. The procedure, therefore, of making the patient cough while the suspected apex is being listened to is of great importance, and the absence of crepitation is frequently decisive in a chronic case of illness as to the presence or absence of tuberculosis.

The symptoms which are produced by phthisis are frequently obscured by tuberculous disease elsewhere, and although symptoms referable especially to the lungs are rarely absent, they may be so slight as hardly to attract attention. This, for example, occurs in tuberculous bone disease or in phthisis following intestinal tuberculosis, in which cases the tuberculosis of the lung is usually of a diffuse form, and spread more or less throughout both lungs.

The physical signs in early phthisis are not infrequently obscured by the presence of chronic lung disease. I may mention two conditions, namely, where an adherent pleura is present on one side, and where general emphysema is present. In the case of adherent pleura it may be due either to a previous simple pleurisy or an apical pneumonia, both of which have healed. Many such patients present themselves, and although they have symptoms referable to the lungs, they are non-tuberculous. The adherent pleura gives rise to the signs of deficiency of movement and deficiency of resonance on percussion, and weakness of breath sounds, and with a patient subject to bronchial catarrh rhonchi may be more numerous on the side of the adherent pleura than on the other side. In the diagnosis of such cases, besides the absence of special phthisical symptoms, such as night sweats and hæmoptysis, examination of the sputum shows the absence of tubercle bacilli, and the results of the treatment of the bronchitis soon enable a diagnosis to be made.

In cases where tuberculosis develops in a patient with well-marked emphysema and rigid chest, the physical signs are frequently very ill-defined, being obscured by the chronic condition of the lung. Thus, although a patient may have definite phthisical symptoms, physical signs in the chest may be extremely doubtful, there being no appreciable deficiency of movement on one side, no alteration of resonance on percussion, and but

little alteration in the breath sounds, which are generally weak, so that in such cases one must rely chiefly on the presence of crepitations on cough, or an increase in the vocal resonance. When, however, the disease advances somewhat, the typical physical signs are discoverable. Phthisis not infrequently develops in patients who are the subject of chronic winter cough. In some cases, indeed, winter cough is actually due to phthisis, which is very slowly progressing, the symptoms being in abeyance during the warm weather, returning at the onset of winter. But in some other cases the history is somewhat different from this, inasmuch as the patient has been for many years the subject of winter cough, with occasional attacks of bronchitis, and presents himself, perhaps, at the early part of the winter, with a history of continuous cough for six or eight months, associated with the symptoms of phthisis. In these cases tuberculosis has developed in patients suffering from emphysema.

In some cases in which there is a strong hereditary taint the development of tuberculosis is very insidious, there being but few symptoms to indicate the large amount of disease which one finds on physical examination. A similar remark may be made as regards those cases of phthisis which develop in chronic alcoholism and in syphilis. Both these conditions I look upon as great aids in the progress of phthisis. In scarcely any condition does the disease progress more rapidly than in patients addicted to the chronic abuse of alcohol, with the exception, perhaps, of some of those cases which follow influenza. And it is a remarkable fact that a large proportion of cases which now attend the hospital date the first appearance of the chest illness from the period immediately following an attack of influenza, which may be either slight or severe. In alcoholism, in syphilis and in influenza the onset of the disease and its rapid progress are rather to be ascribed to the profound effect on nutrition produced by these conditions, whereby the power of resistance to the disease is greatly lowered.

Some cases of phthisis, insidious in origin, are obscured by symptoms referable to the stomach. There may be a period preceding the development of phthisical symptoms in which the patient suffers from dyspepsia in various forms. Sometimes the symptoms may be classed as irritation of the

stomach, associated with hyperacidity. In other cases there is an insufficiency on the part of the stomach, which gives rise to the symptoms usually classed as those of atonic dyspepsia. In still others, however, there may be a period of vomiting, which is severe, and is not dependent, as a rule, on catarrh or inflammation of the organ, but on a condition of irritability. This vomiting, however, is more frequently observed when the phthisis has really developed than before its development, and being, in some instances, coincident with the development of tuberculosis, it masks the symptoms to such an extent that the patient does not complain of cough or expectoration, but complains only of pain after food, and of excessive vomiting. In some cases the vomiting is to be ascribed to irritability of the stomach; in other cases, which are very evidently phthysical from their symptoms, it is to be ascribed directly to excessive cough, brought on by the partaking of a meal. Cases have been described in which a rapid tuberculosis has developed after the mucous membrane of the stomach had been more or less destroyed by corrosive poisons. Such cases are of extreme interest, inasmuch as they help to explain indigestion of food as a factor in the causation of phthisis, namely, by lowering the resistance of the body, owing to the defective digestion and nutrition.

It is evident from what has been said that many cases of phthisis which have an insidious development present great difficulties in diagnosis when the patient is first seen, and it is never wise to make a certain diagnosis when the physical signs are doubtful. It is best to wait for a fortnight or three weeks, and then to re-examine the chest before a decision is arrived at, an examination of the sputum in the meanwhile often clearing up the case.

SUDDEN ONSET OF PHTHISIS.

The development of phthisis is usually insidious, but it may be sudden. For example, in one case of an out-patient—a pale, weakly-looking woman—slight cough had existed for a fortnight, but no expectoration, and no other symptoms of phthisis. There was some fever on the day she was first seen, and the only physical sign that could be detected on two examinations at an interval of a fortnight was a sibilus on cough at the left apex.

She was admitted into the hospital, became delirious the first night, with high fever, and remained in this condition for a short time. After a week or so some expectoration was obtained, which contained tubercle bacilli, and the patient finally progressed as a case of chronic phthisis, the usual physical signs developing at the left apex and becoming extensive. This, however, is not a common mode of onset.

More commonly the disease is ushered in by hæmoptysis. Initial hæmoptysis varies in amount. It is apparently in some cases initiated by a definite effort, but in others it occurs when the patient is quiet, or even on awaking from sleep. It may occur after sudden excitement, or more commonly after some sudden exertion, such as running, lifting a weight, or playing a game, and is thus frequently like the onset of hæmoptysis in mitral disease of the heart. Hæmoptysis is a symptom which is produced by varying causes, the two commonest of which are tuberculosis of the lungs and mitral disease.

It may also be present in renal disease, and in some of the profound anæmias; and, as regards lung disease, it is not an infrequent symptom in basal bronchiectasis following pneumonia. The blood may be brought up in large quantities, red in colour, and frothy, or the mouth may be filled with blood, or blood is brought up in clots—at first red, and becoming darker as the hæmoptysis goes on. There are some patients in whom hæmoptysis occurs and in whom no tuberculous disease develops; and although it is unwise to exclude tuberculosis, especially in young adults, even when no physical signs develop in a period of six weeks or longer after the hæmoptysis, yet there are cases in which the bleeding has appeared to be, so to speak, a pathological accident. I may mention the case of a distinguished member of our own profession—Sir Astley Cooper—who in early life had an attack of hæmoptysis. There was no subsequent development of chronic phthisis, and yet at the advanced age at which he died, a post-mortem examination revealed a fibro-calcareous focus at one apex. Other cases also are not infrequently in evidence in which hæmoptysis occurs, sometimes profuse, and the patient has not developed chronic phthisis. With people of middle age, and beyond—especially those who may be called plethoric—hæmoptysis sometimes

occurs, and is probably, in some instances, of the same significance as an epistaxis. It may occur in the absence of Bright's disease, or of any other of the morbid changes coincident with advancing age.

Another mode in which phthisis commences suddenly is in acute pneumonia. This is purely a clinical aspect of the subject, for pneumonia, pathologically, is in no way related to tuberculosis. It usually leaves no traces of any effect on the lungs, and even when it leads to chronic pneumonia and bronchiectasis, this chronic lesion rarely, if ever, becomes tuberculous. So that it may be considered as fairly certain that even the damage which pneumonia may do to the lung does not predispose to tuberculosis. But it is a clinical fact that many phthisical patients state that they have had an attack of pneumonia. In some instances this has been the commencement of their illness and their serious symptoms; in others it has occurred during the course of the illness. Acute pneumonia may occur in the lung in which there is an old tubercular focus, but there is no regularity as to whether it occurs round this focus or in a quite healthy part of the lung, base or apex. There are cases where there is an old caseous focus at one apex, and at death pneumonia of the opposite base. But the cases which are now under consideration differ from these, and must be considered as pneumonia occurring in patients who have already become infected with tuberculosis, and in whom the acute disease produced great depression of the body, lowering the resistance to the spread of the tuberculosis, so that after the acute pneumonia has resolved, the patient develops definite symptoms of phthisis and definite physical signs at one or other apex. One such case I have seen and verified post mortem, in which a man suffering from chronic alcoholism was admitted into hospital with the symptoms and signs of acute pneumonia at the base of one lung. The sputum was blood red, tenacious, pneumonic in character, but on microscopical examination showed tubercle bacilli. After death it was found that there was an old lesion at the apex of the lung, and in the centre of the pneumonic consolidation was a recent caseous mass, which had broken down, and the contents of which had no doubt been the source of the tubercle bacilli in the sputum. Pneumonia

may also occur in the course of chronic tuberculosis, and may be fatal.

Another mode of origin of tuberculosis of the lungs which is not infrequently observed is, the occurrence of a pleural effusion. In these cases the patient comes not infrequently with a history of an acute illness and stitch in the side, accompanied by a cough and no expectoration (as in ordinary cases of pleural effusion), and on physical examination it is found that at one or other base there are the physical signs of effusion into the pleura: some slight bulging, great deficiency of movement, dulness to percussion, and weak breath sounds, with displacement of the heart's apex beat. As regards the physical signs over the effusion there is no distinguishing point between tubercular cases and those which are non-tubercular. But not infrequently the effusion of tubercular pleurisy is to be suspected from the following facts. The disease is accompanied by night sweats and wasting. It runs a very chronic course, and may frequently require tapping. In acute pleuritic effusion, non-tubercular, as a rule one tapping suffices; the disease usually healing rapidly. The physical signs at the apex of the lung above the pleuritic effusion may be diagnostic of tuberculosis. For example, bronchial breathing with crepitation on cough and after cough may be present, but these may also be present over a lung compressed by a simple effusion. But in some such cases after tapping, when the lung expands, the bronchial breathing and crepitations disappear, whereas in some cases of tubercular pleurisy that I have seen, such physical signs as bronchial breathing and crepitation of the apex remained constant. Or there may be cavernous breathing, which is still more diagnostic of the case being one of tuberculosis. On the other hand, there are other cases in which there is an effusion at one or other base, and no definite physical signs at the apex on the same or other side, and it is only after a period of longer or shorter duration that definite symptoms and physical signs develop. An effusion, therefore, which is slow in developing and still slower in resolution, especially when accompanied, as it sometimes is, by a nocturnal rise of temperature, is to be regarded with great suspicion, even if there are no other physical signs in the chest. In some of these cases an examination of the sputum reveals the presence of tubercle bacilli, which settles the case; but in

some there is no expectoration, and the diagnosis is only made after a time. Similar remarks may be made about empyema, which is sometimes the first sign of the development of a chronic tuberculosis of the lungs. Tuberculous pleurisy is the first sign, then the development of an empyema, and I have seen a case in which this occurred in one side of the chest of a child, and ruptured through the lung, producing an acute caseous tuberculosis of the lung on that side, the other lung being normal.

CLINICAL LECTURE

ON

RUPTURED PERINEUM.

Delivered at University College Hospital, Feb. 18, 1896,

BY

HERBERT R. SPENCER, M.D., B.S.,

Obstetric Physician to the Hospital.

GENTLEMEN,—I propose to make a few remarks to you to-day on the subject of ruptured perineum (of which a case is now in the ward), and of its treatment.

Firstly, as to its frequency. Slight cases are met with quite commonly, and you know that almost every primipara has a little tear of the fourchette after labour; they are as common as the severe forms are rare. By the severe forms I mean such as is presented by this patient, where the sphincter is divided, and the rupture has extended some distance into the rectum.

As regards the causation of ruptured perineum, it may be due to the child's head being too large, or to the head being in a bad position, such as the occipito-posterior position; or it may be due to the outlet of the pelvis being too small, resulting in the head being thrown back, overstretching the soft parts and producing a tear. Again, it may be due to a too-sudden delivery of the child, sufficient time not having been given for the soft parts to distend. Ruptured perineum is very frequently due to the premature application of the forceps. Sometimes a rupture can be

prevented by forceps, but it is more frequently caused by them. The rupture may be due to accidents, to injury of the perineum by foreign bodies; to falling over a bar or over the leg of a chair. The severest perineal rupture I ever saw was caused by the forearm being violently forced into the vagina, during podalic version; in that case the septum was torn for three inches. I have only met with one case in my own midwifery practice of a tear completely through the sphincter, and that was brought about by delivery with forceps through a narrow outlet. In speaking of the rarity of certain affections, it must be borne in mind that hospital cases are drawn from wide and populous districts.

As to the varieties, you may get cases in which the perineum is torn but slightly; this little injury occurs in almost every primipara, and, where the head is large, in multiparæ also; but in the latter cases there is frequently no tear at all. A slight tear of the fourchette is of little importance, and requires no treatment.

You may, not uncommonly, meet with the second degree of laceration of the perineum, in which it is torn for an inch or so, down to the sphincter, but not involving it. The third degree of laceration goes right through the sphincter. You may find degrees of laceration not only from before backwards, but also from above downwards. The mucous membrane of the vagina alone may be torn, while the skin and body of the perineum may escape injury. The wound of the perineum usually begins from the mucous membrane of the vagina, and extends downwards. Or the skin alone may be torn, without the rest of the perineum being affected, or before it is involved. Again, the muscular tissue of the perineum may be torn without the mucous membrane of the vagina being ruptured, or, at least, in only a very small degree. The perineum is generally torn first on the vaginal aspect; then, as the head descends, the muscular tissue, and finally the skin are injured. You may find that after the child's head is born the perineum appears to be intact, or only a slit is apparent, without any considerable laceration; but when the shoulders come down you will be disappointed to find the point of the shoulder will tear the skin, that the bis-acromial diameter has finished the laceration by involving the skin.

Another variety of ruptured perineum I have but twice seen—the so-called “central rupture of the perineum.” This is not an appropriate name, because it does not generally occur in the centre, but more often posteriorly, often involving the anterior fibres of the sphincter. The fourchette remains intact, and a limb of the child or the whole child comes through the perineum. It has been called “perforation of the perineum” by Denman, which is a better name.

In all cases after labour the patient should be examined by inspection to see if the perineum is torn. You will sometimes hear doctors say they have never seen ruptured perineums. A doctor with an experience of 2,000 cases told me so once, and my answer was that he did not look for them. They exist, and the slighter degrees are very common. Ruptured perineum should be treated at once. After the placenta has been delivered and the bleeding has stopped, the perineum should be carefully examined, and when torn it should be sewn up in almost every case. In a rupture of the first degree, stitching is not necessary, but the patient must be kept quiet, clean, and dry. With a tear of the second degree, *i.e.*, more than half an inch in extent, it is necessary to put in stitches. Sometimes the perineum will heal perfectly well without sewing, but it will always heal with stitches, therefore they should be put in. The operation is a simple one. All you require is a needle and three pieces of silk-worm gut, preferably stained with methylene blue, because you can so much more readily see to take them out afterwards.

You may disinfect the silk-worm gut by soaking in 1 in 20 phenol, or 1 in 1,000 sublimate, or by boiling in a test-tube.

Take a long-handled needle made entirely of steel, and put the patient in the lateral, or if you have assistance, or need to give an anæsthetic, in the lithotomy position; then pass the needle just outside the edge of the torn skin towards the posterior part of the tear. Pass it well outward to take a deep hold, and run it round in the tissues, bringing it out at the other side, just catching hold of the skin, but only just. If the needle is inserted too far from the edge of the skin it strangulates the skin, and sets up suppuration. Then thread the needle with silk-worm gut and draw it through, doing this a second and a third time, the third

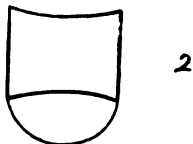
suture not being entirely buried. These three sutures are sufficient to close the wound. Iodoform should then be applied to the part, and if the injury be deep, a catheter should be passed for the first three days. In the case of a tear of the third degree, it is still advisable to stitch up the perineum at once if the patient is in a fit condition. In some cases, this step cannot be borne by the patient, owing to shock or hæmorrhage, and it is then advisable to wait for a few hours, but in almost all cases the operation should be done immediately. If this be not done within a few hours, it is generally better to wait for at least six weeks, until the genital tract has involuted. But you may do it at any time, even years afterwards, with perfect success and the details of operation are the same, whatever the interval.

I shall now describe the operation which I shall do on this woman. When the tear goes through the sphincter, the operation is somewhat different from the one I have just described. If you examine this patient on the couch, you will discover the septum between the vagina and the rectum, the lower edge of which is scar-like, and at the outer part of which are some little puckering, which are really due to the retracted ends of the sphincter ani, and in freshening this surface in cases of old standing, it is of very great importance to cut across the divided ends of this muscle. First, take a pair of sharp-pointed angular scissors, and remember that for all plastic operations the instruments must be sharp. With the scissors split this septum, not cutting the mucous membrane of the rectum, but severing this puckered part by curving the incision so as to divide the ends of the sphincter. From the ends carry the incision

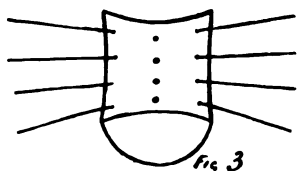


forwards, towards the labium minus to a variable extent. Some make the incision H shaped, but by making the transverse incision well-curved (see fig. 1), the posterior limbs of the H are avoided.

Then peel up with the scissors the flap, slipping the scissors under it, taking as little away as you can, peeling up the skin of the vagina as far as necessary. It can be done more quickly with scissors than with a knife. Then you get a surface which is shaped thus:—

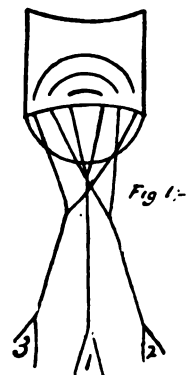


the lower side being hollow. The raw surface is then ready for stitching, though you may need to apply forceps or hot sponges, or (rarely) a ligature to bleeding vessels. In order to close the wound—to repair the rectum, sphincter, and perineum—two (of many) methods may be adopted. One is termed the “Lawson Tait” method (see fig. 3),



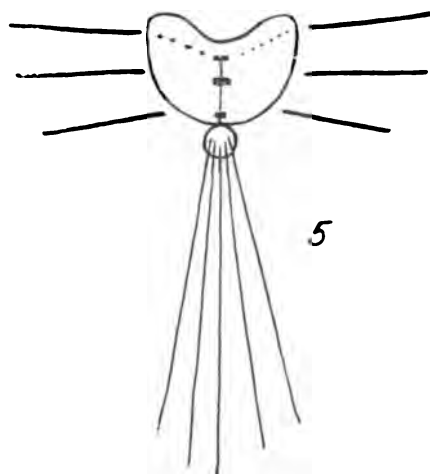
which is simple, but less efficient for extensive tears than the other I shall describe. By the Lawson Tait method, the needle is passed within the edge of the skin, without taking up the skin, then it is buried almost completely beneath the raw surface, except that it just appears in the middle of that surface. The needle is threaded with silk-worm gut, and three or four sutures are passed, none going through the skin or appearing in the rectum. Then they are tied, and, if necessary, some superficial sutures are put between the deep perineal sutures. For slight tears through the sphincter this operation is quite successful, but is less efficient for deep wounds than the method I am about to describe to you, and to perform immediately. The surface having been prepared as before, and shaped as in fig. 2, we take a small half-curved wire needle with two eyes, threaded with fine silver wire, then with a needle-holder (of which Martin's is the best and Spencer Wells's almost as good). This needle is inserted just at the cut edge on the right of the middle

line (fig. 4), a short distance from the apex of the tear, and buried a quarter of an inch, and



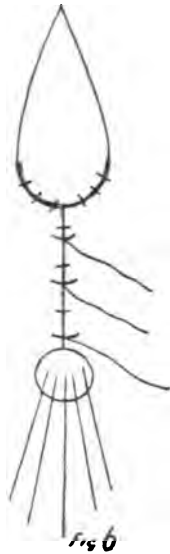
afterwards brought out on the raw surface. Then take it over and insert it to the left of the middle line, a quarter of an inch from the cut edge; after which bury it and bring it out exactly at the cut edge (see fig. 4). Having inserted this wire, you must use the wire-twister to twist the wires up tightly, but not sufficiently to strangle the tissues, only to bring the edges into apposition. One-sixth of an inch below this wire, insert another wire (fig. 4), similarly, twisting that up also, repeating until you have finished at the sphincter. You will then have a bunch of wire hanging out of the anus.

Now you have left a simple second degree rupture of the perineum (fig. 5), which you treat



as before, with a long-handled needle, with which you just take hold of the skin; but in burying it in the tissues be careful not to cross the loops of

the rectal wires. If you do cross them, and infection occurs from the rectum, the whole wound may become infected. Having sewn up the perineum, you have the flap that you have raised lying on the top of the perineum. Generally it



is recommended that nothing be done to that flap, but, from experience, I know that if you want a perfect union by first intention, it is better to trim off any little prominent points in this flap, and to carefully suture, with horse-hair, the flap to the anterior border of the new perineum, by means of Hagedorn's needle and Pozzi's needle-holder.

Now, that is an operation which is uniformly successful when carried out as I have explained in cases of complete rupture; by success I mean that it unites the sphincter and the perineum, thus restoring to the patient the power of retaining fæces and flatus.

In the after-treatment we generally give a suppository of morphia to lessen pain, and the patient has the knees loosely tied together to prevent her widely separating the legs. A catheter is used every six or eight hours for the three or four days after the operation. The best catheter is, I think, a glass one, such as I have had made recently, which will go into a test-tube, and can thus be readily rendered perfectly aseptic and non-irritating by boiling. It is very important that the bowels should be opened on the third day by a saline, and be kept open regularly afterwards.

The old-fashioned plan of keeping the bowels confined for ten days was responsible for many failures. The wound must be kept clean and dry and iodoform wool should be applied to it.

As regards food, I would only advise you not to let the patient eat food which contains any indigestible remnants, such as oranges. I have known one of the stitches suppurate through the catching of an orange pip in one of the rectal wires; and we had a case where a small flake of mutton-bone was swallowed and was caught in the wires.

The deep perineal sutures are taken out on the ninth day, and here you will find it useful to have the silk-worm gut coloured blue. The other sutures can be taken out at any later period, say, on the fourteenth day, and they are withdrawn in this way:—Wash them with a stream of water, take hold of the lowest of them with a Spencer Wells, and pull until you see the loop. If necessary take up a small bit of wool in Spencer Wells's forceps, to wipe the dirt and blood from the loop, and you can thus cut the loop and withdraw the wires. Occasionally, but rarely, it may be necessary to pass a rectal speculum in order to withdraw them.

The operation which I have described to you takes longer than that known as the Lawson Tait, but it has succeeded in my hands several times when the other operation has failed in the hands of others. Therefore I believe it is more generally applicable than the Lawson Tait; if performed directly after labour, the operation is essentially the same as that which I have described to you. Of course, no freshening is necessary; an anæsthetic is only necessary for the cases in which the tear is of sufficient severity to be classed in the third degree.

In every midwifery case the perineum should be supported with the right hand. The too rapid propulsion and extension of the child can be prevented by this means, and by the pressure of the forefinger of the left hand on the head of the child. If this practice be observed in every case, and a sufficient time be allowed for the perineum to distend, complete rupture of the perineum will hardly ever be met with.

The cases of so-called central rupture of the perineum should have the band in front of the tear divided, and then be stitched like an ordinary case

HERPES ZOSTER.

Notes from the Clinic of

THOMAS BARLOW, M.D.,

At the University College Hospital, January 17, 1896.

THE patient exhibited was a little girl with chorea. She had been having three minims of liquor arsenicalis thrice daily for some weeks, there had been no stomach trouble, and no feeling of sickness or loss of appetite, and her movements were much improved, but she had been brought on that day to show a little crop of herpes on the back. It is quite certain that sometimes herpes zoster follows upon the administration of arsenic, and it may occur quite apart from any gastric features. In the effects from gastric disturbance are placed the "silvery" tongue, the nausea, and vomiting and pain over the stomach, and diarrhoea, which constitute a distinct group, and with them it must be remembered conjunctival injection and smarting in the eyes, and occasional slight albuminuria. Here is a different group in which arsenic appears to have a specific effect on the peripheral nerves. In this particular case, it was a neuritis of the intercostal nerves, and it is enough to say that herpes zoster may occur whilst people are taking arsenic, and it is not at all necessary that large doses should be taken, for Dr. Barlow knew a lady who took one-fiftieth of a grain twice a day, and yet after a week she had an attack of herpes zoster; she left the medicine off and the herpes disappeared, and on resuming the medicine she had another attack of zoster. It is a curious thing about zoster that it very rarely occurs a second time in the same person, but there are several cases on record where people have taken arsenic, and have suffered from recurring herpes. It is well that it should be remembered in connection with this subject, that there have been several examples recorded of grave peripheral neuritis affecting the limbs, with extreme atrophy and motor and sensory paralysis, in patients who were being treated with arsenic. But in these cases, so far as memory serves, the medicine has been given either in large doses or over protracted periods, whilst in the occurrence of herpes zoster we have to recognize, as has been pointed out, that the dose given may be small. It would seem that in the proneness to get

zoster whilst taking arsenic there is a special idiosyncrasy of the individual, such as we find in relation to toxic effects of some other drugs.

A more common result of the medicinal use of arsenic, with which you ought to be familiar, is the pigmentation of the skin which sometimes occurs.

If the cases of St. Vitus' dance are watched in which arsenic is administered for a few weeks, in a certain number you will find the skin of the axillæ, the bends of the elbows, the popliteal spaces, and the front of the abdomen becoming dark, and if there are any old cicatrices on the body the skin around them becomes dark. This pigmentation rapidly clears up when the administration of arsenic is stopped.

REVIEWS.

Phonographic Outlines of Medical Terms.

Issued by the Society of Medical Phonographers. Small 8vo., 70 pp. (London: Sir I. Pitman and Sons, 1, Amen Corner.)

Price 1s. 6d.

The increasing number of medical shorthand writers has made necessary this list of shorthand outlines for about 2,500 terms. It should be a veritable *vade mecum* to medical phonographers; and with it many of the trials of the inexperienced writer will disappear, and none need now be afraid to take up the study of shorthand for medical use. In the introduction the use of contractions is discussed, and some useful suggestions are made on those of special use to the medical writer. Then follows the list, arranged alphabetically. The outlines are, we understand, those which actual use has proved to be good, and that, therefore, for *succussion*, where, in violation of phonographic rule, the circle and the large hook appear on the same side of a straight letter, is an oversight. We think, also, that the double-length *n*, might with advantage have been used for the series of words beginning at *enteralgia*, whilst the omission of *choroidal*, *excoriation*, *hymen*, *retinoscopy*, and *phylctenula*, scarcely detracts from the first issue of a list which is wonderfully complete, thoroughly practical, and excellently printed.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers. Small 8vo. (London: Sir I. Pitman and Sons, 1, Amen Corner.) Price 4d.

In the number for January with which the new volume commences, the adoption of a larger page and bolder shorthand is a great improvement, whilst the size of the magazine is not too large for the pocket. Dr. Bird's paper on the use of ice-bags in facial erysipelas shows that ice is more comforting than other local applications, and that patients recover equally well. A 2 per cent. ointment of monochlorophenol is first applied on a mask of lint, and upon this a specially made ice-containing helmet, with separate face and forehead pieces, is placed. The value of a useful contribution would have been enhanced by the addition of the name of the maker of the ice-helmet, and mention of the hospital to which Dr. Bird is attached. Dr. Huggard, of Davos, divides climates into those where the heat demand is large and small, and he points out that such a thing as a dry, equable climate does not exist. The dry cold of the higher Alpine resorts is suitable for robust subjects apart from their local disease, and for others, who, not robust, have powers sufficient to respond to the increased heat production the climate demands. Those with feeble circulation, poor digestion, and small lung capacity, will find the climate depressing and harmful, and the maximum of good is to be expected in cases where moderate pyrexia accompanies localised disease of one lung.

A previous diagnosis of cerebral hæmorrhage in a case of poliomyelitis affords Dr. Gowers an opportunity to elucidate another of his instructive problems in practical diagnosis. Dr. Gowers points out that in cerebral hæmorrhage the leg is seldom paralysed more than the arm, whilst in poliomyelitis the muscles waste rapidly, and after ten days their faradic irritability will be diminished or lost. The lesson of the case is that the electrical reaction of the muscles should be ascertained in all parietic conditions.

We are told that the Society of Medical Phonographers now numbers more than 200 members, and that there are six manuscript magazines circulating among the members, in addition to the excellently lithographed publication before us.

In the February number Dr. Huggard concludes his notes on the selection of climate in phthisis. South Africa is unfitted for those with feverish tendency, and those fastidious in the matters of food, accommodation and society. It affords a permanent place of residence for those who can rough it a little and earn their own living. The Rocky Mountain resorts are not so cold as the Alpine, and are better suited for cases of low vitality. The Riviera is suited to invalids who have not the vigour to respond to the cold of the Alps, and the same applies to the Canaries, Egypt and Madeira; whilst the English South Coast suits many even too ill to send abroad, and cases with digestive troubles and diarrhoea are better there than on the Continent. All cases with high degrees of pyrexia—over 102° in the afternoon, not falling to normal in the morning—are best at home, and no migration to a health resort should be thought of till a less active condition is reached. Hæmoptysis in the early stages is no contraindication, but hæmorrhage from large cavities only shows a tendency to recur at the high altitudes. Night sweats usually cease in a cold dry climate, diarrhoea should be an indication for the patient to stay in England, and catarrhal affections of the larynx and pharynx, which are apt to be irritated by the excessively dry air of the Alps, do well at Mentone and Orotowa, if a sea-voyage does not seem desirable.

Dr. Gee draws attention to a little understood malady—epidemic cerebro-spinal meningitis, too often, it is to be feared, in practice confounded with meningitis. It is a specific infectious disease, due most commonly to the pneumococcus. The symptoms may be divided into three groups: (1) Those of an infective fever, lassitude, headache, picking of the bed-clothes—a characteristic symptom; eruptions like typhoid spots, limb pains and swollen joints. (2) Those relative to the brain, like meningitis, delirium with concurrent headache, and not, as in typhoid, following the headache, infrequent and irregular pulse, convulsions and coma; and (3) Symptoms relative to the spine, and these, which are characteristic of the disease, comprise stiffness of the muscles, especially of the back of the neck, of the back and limbs. The head is rigidly thrown back. The onset of the disease is always sudden, with a rigor in adults, a convulsion in children, and in all with vomiting. The diagnosis between typhoid and typhus and the disease is not always easy. The treatment consists in seeing that the patient is kept quiet and well nursed, and Dr. Gee recommends belladonna and iodide of potassium as the special drugs to be administered, whilst the application of ice to the spine is found to give much comfort.

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 18, 1896.

TWO LECTURES ON ADULT ANTERIOR POLIOMYELITIS LECTURE II.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, May 22, 1895,

BY

W. R. GOWERS, M.D., F.R.S.,

Physician to the Hospital, and Consulting Physician to University College Hospital.

IN every case of disease, the diagnostic problem consists, first, in discerning the symptoms, in making out, as it were, the words of the message which the disease presents to us. Then we have to see what sort of a sentence the words form, by putting them together, and next to read its meaning. You will remember that, at our last meeting, we investigated the character of the symptoms in the patient who is now before you, who has wasting of the muscles of the right leg. We observed the symptoms carefully, and I think we need not go over them again. You will remember that the muscles are much wasted, faradic irritability is lost, voltaic irritability persists, although it is less than normal. The reflex action is lost, alike the plantar reflex from the skin and the knee-jerk, loss of which indicates the loss, not of a reflex action from the tendon, but of the reflex action from the afferent muscle nerves. We saw that the persistence of sensation prevents us referring any share in the production of that loss of reflex action to the sensory fibres.

To-day we have to consider what those symptoms signify—what is the meaning of the sentence that is formed by their combination? In other words, having discerned the character of the symptoms, we have to ask “where is the disease?” and “what is the disease?” I mentioned some of the indications of the position of the disease, but I may remind you of them, and point

out what their significance is as regards localization. I explained that they signify disease in the lower segment of the motor path—the segment which consists of the nerve cell of the anterior horn of the cord—the branching processes from it, which, in the spongy grey substance, meet those from the pyramidal fibre, by which the impulse is conveyed from the brain. It includes also the branching processes which meet those from the posterior roots, and likewise the great axis-cylinder process which passes down to the muscle, and there, dividing, ends upon the muscular fibres. The loss of faradic irritability means loss of the influence of the nerve cell on the motor nerve fibre, or it means some influence on its nutrition more potent to damage than the vital influence can counteract. The latter is the mechanism at work in multiple neuritis, but it is with the former we have now to do. The loss may be due to disease in almost any part of this cell-fibre element. The muscular fibre itself does not respond to the extremely brief “induced” current of electricity. That which we call the faradic “current” or induced “current,” merely consists of a rapid succession of shock-like currents, each of which is so brief that the low sensitiveness of the muscular protoplasm cannot respond to it, although the more sensitive nerve structure can respond. When, in health, the faradic current is applied to a muscle, contraction is caused by stimulating the endings of the motor nerves. That which complete loss of faradic irritability shows is the loss of the excitability of these nerve endings.

Even when the nerve fibre itself, between the cell and the muscle, is intact, and only the extremities of the terminal ramification are affected, there is complete loss of faradic irritability. The nerve structure has a far higher degree of excitability, due to a more elaborate, more delicate, more complex constitution, than the muscular protoplasm, and alone can respond to so brief a stimulant. If we apply even voltaic electricity to the muscle in health, the contraction is due to the stimulation of these more sensitive nerve fibres. If the functional power of the nerve ends is

abolished by curara, the nerve impulse excited cannot reach the muscle. There is the same loss of response to faradism as if the whole motor nerve were degenerated.

The stimulation by the voltaic current is effected by interrupting the circuit; it is due to the sudden change from no current to current, and *vice versa*, or to a sudden increase or decrease. This, effected by the hand, has a far slower influence than that of the induced current. The less sensitive muscular tissue can respond to it, although not so readily as the normal nerve fibres. In health, therefore, the latter are first stimulated. But if the function of these nerve fibres is abolished from any cause, the muscular protoplasm, unable to respond directly to the very brief faradic shock, can yet respond directly to this slower stimulus of the change in the voltaic current. Hence, when the nerve endings are degenerated, we still find this contraction from voltaism.

Thus, the loss of faradic irritability of a muscle proves only loss of the nerve endings, it may depend upon disease in any part of the segment, in the cell or the fibre. This fact is the first point in our present problem. This wasting which you see to be so conspicuous, with flaccidity of the limb, with loss of knee-jerk, with loss of plantar reflex, must be due to disease of the motor nerve cell of the grey anterior horn, or of the fibre proceeding from it, or of the extremity of the fibres. Can we form an opinion in which of these positions the disease is?

There are four places in which such disease specially occurs, viz.: (a) the grey matter, (b) the nerve roots, (c) the nerve fibres in their course, (d) the nerve endings. The whole leg is affected, and yet only the motor fibres have suffered. The motor and sensory roots are so near together that it is impossible to conceive a lesion such as this, so extensive in its range, should affect all the motor roots for this one limb, and leave the sensory roots untouched. That fact alone enables us to exclude the nerve roots as the seat of the disease. Could it be in the nerve trunks in the limb? We should then have the same difficulty; in the nerve trunks the fibres for sensation and for motion run together: and the fact that sensation is not involved excludes also this position.

Can it be disease of the nerve endings? We

have learnt that many toxic agents affect the peripheral extremities of the nerves, and may affect alone the motor nerves, and leave the sensory nerves unchanged. We have examples of this in curara, and in some metallic and other poisons. But those poisons have to reach the nerve endings through the blood, and therefore they could not have an influence on one leg only; any poison capable of influencing the nerve endings, and causing peripheral neuritis, would obtain access to left leg in the same degree and intensity as the right, and there would inevitably have been an approximately symmetrical paralysis. Remember the all-important fact that symmetry is the great characteristic of toxic influences, and peripheral neuritis we know only as an effect of toxic influences. So that, by the process of exclusion, we are left with only one place to which we can refer the disease—the anterior grey matter of the cord. There alone is it conceivable that a morbid process might exist, might produce these effects, in this limitation.

But what is the morbid process? You know that symptoms, by their grouping, tell us where disease is; the mode in which they come on shows us the general character of the disease. In some cases we have to rely upon that indication only, with the help of what we know of the relative frequency of the different morbid processes; but in some cases we are also helped by being able to perceive a cause for one of these morbid processes. The mode of onset, then, is the great guide to the pathological diagnosis. A morbid process which occurs with literal suddenness must be of vascular origin. It must be due to the rupture or occlusion of a vessel. A morbid process which occurs acutely, not suddenly, is occasionally, but rarely due to a vascular process. Its common cause is inflammation. A morbid process which develops subacutely or subchronically may be inflammation or growth. One which develops in a chronic manner may be chronic inflammation, or a growth, or sometimes a degenerative process, such as may be, and often is, included in the term "chronic inflammation." A morbid process which develops in a very chronic manner must be either a growth (if local) or a slow degenerative process, such as some authorities term "chronic inflammation," but is better regarded as degeneration. If it is actually sudden, the lesion

is vascular; if the onset is in a few hours, a few days, or a week, it is probably inflammation. If in a few weeks, it is inflammation, or possibly a growth. If in a few months, it is inflammation or a growth. When some years pass before a considerable degree is reached, it is a growth or degeneration. In this, however, is included the slow forms of sclerosis, that is, of connective tissue overgrowth. Commonly secondary to wasting of the nerve elements, it is sometimes primary; then it is random, and on account of its distribution may perhaps be regarded as at least allied to very chronic inflammation.

In this case, the symptoms occupied one week in their development. Eight months ago they began with pain across the lower part of the back, passing down to the groins and thighs. The pain lasted only six days, and as it came on the right leg became weak. When the pain ceased, the right leg was powerless, and powerless it has continued since. Those are all the available facts regarding the onset, but you will see that it is the onset characteristic of inflammation. We know that the anterior grey matter of the cord is a frequent seat of inflammation in children, but seldom at this time of life. Still, it does occur, and such acute inflammation is not met with in the nerve roots, nor is it limited to the motor nerve fibres of one limb. If such acute inflammation occurs in the nerves outside the cord, it is due to meningitis, or to an acute inflammation of the sheaths, and it affects alike the sensory and the motor fibres, or it is due to an acute toxic influence on the nerve endings, and then is bilateral. Thus the indication afforded by the mode of onset harmonises with the indication afforded by the symptoms, that it is an affection of the anterior grey matter of the cord. We must conclude that the patient has had an unusual form of acute or subacute poliomyelitis, affecting the anterior horn throughout the lumbar region on the right side. It was most severe, and very little improvement has yet occurred, although eight months have elapsed.

I said that this inflammation of the anterior horn is rare at this time of life. It was formerly thought to be not uncommon, because cases occur with similar symptoms, but with bilateral symmetry. We now know that these subacute and subchronic forms of symmetrical "poliomye-

litis" in adult life, which until about ten or twelve years ago were described as such, must have been cases of peripheral neuritis. This case is not one of this class. His symptoms are not symmetrical. This man must, therefore be the subject of adult poliomyelitis.

I have dealt thus at length on the steps of the diagnostic process, because it is an example of that which is frequently needed, in which we have to combine the character of the symptoms, and the mode of their onset. The process we have often to adopt, but it is unlikely that any one of you has seen a case before such as this.

You will often meet with cases which you find it difficult to make out with sureness. But if you deal with such cases in the way I have just dealt with this, if you consider what the symptoms are, what position of disease their combination indicates, what character of lesion their onset suggests, and whether that lesion is probable or a possible one, and if you trust every step you take, you will arrive at a correct diagnosis in the vast majority of cases, whether they are unfamiliar to you on account of your ignorance, or unfamiliar to you by reason of their absolute rarity. I think each one of you may feel that if this case had presented itself for you to make the diagnosis, if it were possible for you to have heard this lecture and absolutely forgotten it, and the lecture merely left the result it should produce on your diagnostic tendencies, you would have arrived at the diagnosis which I have just given you. Had you done so, you would have formed a correct diagnosis in a case the precise like of which I have never seen before; I have never seen a case of adult poliomyelitis involving the whole of the grey matter on one side of the lumbar enlargement. I have had to make a diagnosis in the same way as that you would have had to employ. You may have to apply the process to a case in which I might seem at once to jump to a correct conclusion; but the seeming jump would really be a rapid passage through the same diagnostic process, and I should have to depend, as much as you, upon careful reasoning. If you grasp these facts, and proceed from symptoms to their meaning, throwing aside all types, and (more important still) all names, you will find that the number of cases which will baffle you will be very small. I have said all this before; you will find it all in print.

But I think that it is good for nine out of ten students to have that which is important impressed on them twice; and that this is necessary in the case of three out of every four, while it is inadequate in one out of every three.

Eight months after the onset of any acute inflammation—what is the condition? Consider what would be seen in a part accessible to you. We are ready enough to use analogy in theory, but strangely indisposed to make the really legitimate use of it—to endeavour to see with the mind that which is like a process that can, elsewhere, be seen with the eye. In a visible structure, the seat of such inflammation, after nine months, there will be some absolute destruction, some damage short of destruction, yet most of the tissue elements that are not destroyed will have recovered. But, recovery of function in the central nervous system follows, often at a long interval, apparent restoration of structure; the symptoms of loss continue for a much longer time than corresponds to the renewal of the structure. Structure may be restored, degenerated fibres may regain continuity, and yet conduct imperfectly; the process of conduction increases in degree in those elements whose continuity is restored, increases for many months, and often for years. So that in this case, the spontaneous improvement, which is due to the regeneration of damaged structures, may be considered as still far from being at an end.

Yet there is another side to be considered. We have here to do with nutritional centres, and not with mere conduction. We have to do with the nerve cells that govern the nutrition of the fibres that conduct. In the former, the capacity for recovery of structure is less than in the latter, except by function. It is probable that the improvement that has yet to come is small compared with that which has already come; and, alas! that which has already come is also small. Moreover, when the effects of inflammation are over, and cicatrization has occurred, the condition is practically beyond our influence. Use here your power of "reasoning by analogy," and consider the state of a destructive acute inflammation of the skin, that is, ulceration. Consider the condition eight months after an acute process which rapidly went on to cicatrization. Transfer your conception to the grey matter of the cord. Cicatricial tissue will subserve many of the functions

of our cutaneous envelope, but what functions of the anterior grey matter of the spinal cord can it subserve, according to our present knowledge? We do not know, however, at this date, that all the loss of function means destruction, that is irreparable. I will bring this subject before you another day. But some further conducting power and nutritional influence on the muscles may yet be regained. We can and should, indeed, sedulously maintain the irritability of the muscles by the use of electricity, to lessen the secondary processes of degeneration in their structure, and to make them able to respond better to such nerve impulses as the slow return of function may permit.

I have known instances in which, long after disease of the lower segment of the motor path had become stationary—when there has been no return of power, and there has been great wasting—the evidence of benefit to be distinct. The first application of voltaic electricity failed to obtain any response, but after it had been applied for a few days a distinct reaction was obtained; that reaction increased and extended until, in the course of two or three weeks, there came definite power of voluntary contraction, and the improvement went on and on. I think that if the muscles had not been thus stimulated, voluntary power would not have returned.

Of the influence of electricity on the nerve structures themselves we know little, and nothing regarding its influence on the cord. Only one agent seems to have a definite influence on the recovery of function, and therefore of nutrition, where recovery is possible. It is strychnia. In progressive muscular atrophy its effect does not admit of question. But for this, strychnia is incomparably more useful when injected beneath the skin than when given by the mouth. We may conceive that the difference is due to the more sudden contact of the molecules of the agent with those of the living tissue that is susceptible to them. You know how great is the difference in the effect of a mechanical impulse of given force, if it is spread over an hour or compressed into a minute. A "foot-pound" would be wasted if spread through five minutes, and yet it would do a good deal of damage to many things if it were applied in a second. This is, of course, only an analogy—an allegory, if you like. But

these semblances, though sometimes only such, sometimes cover realities.

The nitrate of strychnia is the best for the purpose, and may be given in a dose of $\frac{1}{80}$ gr., increasing to $\frac{1}{25}$ once a day. It is not wise to increase the dose, although some patients in our own profession, to whom it has done good, have felt its beneficial influence to such a degree that they have carried the injections up to $\frac{1}{20}$, $\frac{1}{15}$, and even to $\frac{1}{10}$ gr., without inconvenience. But $\frac{1}{20}$ sometimes causes enough inconvenience to make me advise you not to go quite up to it.

Massage to the muscles, especially upward rubbing, promotes the movement of the lymph and of the blood, and by the mechanical effect upon the muscles, may stimulate a little the contractile function of the muscles, and thus help their nutrition. It must have, at least, an influence in the right direction. More than that cannot be said, at least, with confidence. Remember that we are not now concerned with the effect of massage on general nutrition, but on its local and direct influence on muscles which are wasted from disease of their nerves. Valuable as is this therapeutical measure, the results on which high estimates are based are seldom free from many sources of fallacy. But, finally, remember this; whenever the will can excite a fair amount of contraction; this, the normal stimulus to overgrowth, has an influence incomparably greater in degree, and more direct in action, than any extraneous agent we can occasionally bring to bear upon the muscles.

CLINICAL LECTURE

ON

A CASE OF ACUTE GENERAL PERITONITIS,

TOGETHER WITH REMARKS ON THE ADVISABILITY OF OPERATION FOR INFLAMMATORY CONDITIONS ARISING IN CONNECTION WITH THE VERMIFORM APPENDIX.

BY

W. HALE WHITE, M.D., F.R.C.P.

MARY H., æt. 22, was admitted on January 17th, 1896, for abdominal pain and distension, with much collapse. On inquiring into the

history, we found that her health was usually good, but that two years ago she had pains in the abdomen, and was very queer for a month. Every now and again since she has complained of similar pains.

The present illness began a fortnight ago with internal pains. These were very severe, and were always referred to the right iliac region. She was very sick for the first three or four days, and since then the nausea has been so marked that she has only been able to take a very little milk. The pain was so bad that she went to bed, but as in three days she was no better, she got up and walked to see a doctor, who ordered hot fomentations. She went back to bed, and has remained there almost constantly, only getting out occasionally. No doctor has been attending her. Her bowels have been constipated during the illness, and consequently she has frequently taken purgatives. The bowels were open last on the 16th instant. The pain has gradually got worse, and has spread over the whole abdomen. On the day of admission she walked up to the hospital, about a third of a mile from her house. The journey took her two hours.

On admission.—She is much collapsed. Her hands are cold and clammy; her face pale, with bright eyes. Her pulse is small, soft, and rather running, 120. Her temperature was subnormal when she was first seen, but after she was put to bed and made a little warmer, it was 101.2. The tongue was slightly furred and rather dry. The abdomen was uniformly and considerably distended. It was so tender that little could be made out on examining it, but it was resonant all over, except for an area of dulness in the right iliac fossa, and here the resistance was, if anything, more than elsewhere. The breathing was shallow, quick, and entirely thoracic. On examining *per vaginam*, a considerable resistance could be felt in the region of the right broad ligament.

We ordered a grain of opium every two hours for the first six hours, and after that it was given as often as was necessary to keep her thoroughly under its influence. Hot fomentations were applied to the abdomen, and she was ordered two ounces of milk every hour. At night she was much troubled by a hacking cough, which was unaccompanied by any expectoration.

January 18th. This morning she is cold, sweats

considerably, and is still in a collapsed condition. The pulse is rapid, small, feeble and running, so half a teaspoonful of brandy was ordered every two hours, and this was in the afternoon increased to a teaspoonful and a half. The temperature in the early morning again fell to subnormal, but in the afternoon it rose to 101. The abdominal pain and tenderness are much less, and she remains quiet, but the distension is unaltered. In all respects she became worse as the day went on, so strychnine was given subcutaneously, and in the afternoon as continuous vomiting began, feeding by the mouth was stopped, and she was fed *per rectum* with enemata and nutrient suppositories, and the morphia was given subcutaneously.

January 19th. The patient is still very collapsed. This morning her temperature was 96, and the pulse was so weak that it could hardly be counted. In the afternoon she was very restless, and the vomiting was very frequent; the stomach was washed out, and after this the vomiting stopped. It was directed that she should have no more morphia. She sank and died about 8 p.m.

When we first saw this patient, the history of abdominal pain, tenderness and distension, the vomiting, and the constipation, pointed to acute peritonitis. When we examined her, the uniformly distended immobile tender abdomen confirmed this view, which well accorded with the state of the pulse, her furred tongue, the reflex cough, and her expression. Just as there are two varieties of severe acute pleurisy or pericarditis, namely, those in which there is a large amount of lymph, and little or no fluid, and those in which pus is more marked than lymph, so you meet with severe acute general peritonitis, in which there is a large amount of thick lymph binding all the intestines and organs together, although but little fluid is secreted, and severe acute general peritonitis, in which there is not much lymph, but a large quantity of pus, which always contains numbers of micro-organisms, most of which are bacillus coli communis. These two varieties merge one into the other, but in extreme cases the distinction is well marked. It appeared probable that here we had a large amount of lymph with very little fluid, for not only were there no physical signs of fluid, but acute suppurative peritonitis in a person in previous good health, is usually due to the perforation of some viscus, and then the patient is taken seriously ill with severe

pain, and hardly ever lives a fortnight. In passing we may notice that often when suppurative peritonitis terminates a long-standing disease, in which the patient is very weak, as, for instance, when it follows perforation at the end of a long attack of typhoid fever, or perforation in the course of malignant disease of the bowel, its presence may be quite unsuspected during life. During the last year there have been three or four instances of this in my wards. Having decided that this patient had acute general peritonitis, which was chiefly non-suppurative, we next had to think of the cause of it.

In women, the three common causes are disease of the pelvic organs, disease of the vermiform appendix, and a perforated gastric ulcer. We quickly put the last out of court for there was no evidence of it. The onset of the disease was not sudden enough, and the local signs in the right iliac fossæ pointed to one of the other causes. The dulness and increased sense of resistance here, together with the patient's statement that she first felt pain in the right iliac region pointed to a peritonitis, at first local, and soon becoming general, which had taken its origin in the appendix. The thickening felt in the neighbourhood of the right broad ligament made us, however, hesitate, for it was difficult to say whether the peritonitis had taken its origin in the structures in the right half of the pelvis (probably the Fallopian tube), and had spread chiefly in the direction of the right iliac fossa, giving the signs there, or whether the trouble having started in the appendix, had spread downwards into the pelvis so that it revealed itself on examination of the vagina. On the whole, we thought that the history of previous attacks rendered appendicitis the more likely of the two.

I have met with three or four cases in women in which the diagnosis between the two conditions gave rise to great difficulty. For instance, only quite recently I have seen a case in which the fact that an attack of appendicitis occurred about the time that a patient miscarried led to a wrong diagnosis, as some thickening could be felt on vaginal examination. I have also seen a case in which appendicitis led to an abscess in Douglas's pouch, which burst into the rectum, and I know of an instance in which after a severe attack of appendicitis the vermiform appendix became adherent to the ovary, with the result that at each

menstrual period the patient had a severe attack of pain and rise of temperature, nearly resembling the first attack of appendicitis, and Dr. Cullingworth has recorded (*Lancet*, July 9, 1892), a case in which suppuration in the ovaries was due to contamination from an inflamed appendix.

As soon as we had come to the conclusion that this woman had acute peritonitis the question presented itself, should we operate. We decided not to do so, for the following reasons:—

Operations in the early stage of peritonitis soon after the perforation of a gastric ulcer have often proved successful, but with this exception, laparotomy for acute peritonitis is one of the most fatal operations in surgery. It would certainly not have been justifiable in the present case; the patient was cold, collapsed, and almost pulseless from the very beginning. Then, too, you will remember that we thought this patient had chiefly plastic peritonitis, without much pus. Now, this is the only variety of acute general peritonitis that can recover under medical treatment, therefore, slight as was her chance of recovery, it was clearly better without an operation. The acute suppurative form is necessarily fatal, if left to itself; therefore, if the diagnosis renders this quite certain, and the patient is in such a condition that you think she may survive the cleansing of the abdominal cavity, as this is the only way life can be saved, you may try it; but remember two things—you must tell the friends that it is one of the most fatal operations in surgery, and you must be absolutely sure you are dealing with the suppurative, and not the plastic form, in which the patient stands a better chance without operation, and the adhesions render it almost impossible to cleanse the cavity. I have never seen a hospital patient suffering from acute suppurative peritonitis whose condition was good enough to make it justifiable to try, but on three occasions in private practice, the patient, having been well treated from the beginning, the attempt has been made to save life by washing out the peritoneal cavity; all three were fatal.

If in a case of peritonitis you decide not to operate, you must give plenty of opium. I never saw too much given. The best way is to give a grain of opium in the form of a pill every two hours till the pupils are gently contracted, and then diminish the dose so that you keep the patient in this condition till the symptoms begin to subside.

Be guided by the pupils rather than the respiration, for the peritonitis alone often makes this difficult. Never mind the constipation; the quieter the bowels are the better. When the patient is well again a simple enema will overcome this. If she is sick you should discontinue the opium, and give morphia subcutaneously—in fact, many people prefer this to opium; but it always seems to me that some other of the constituents of opium may be doing good for aught we know. It is often very useful to combine a quarter of a grain of extract of belladonna with each grain of opium. It helps to keep in check the disagreeable symptoms of restlessness and vomiting which opium produces in some people. Thin flannels wrung out in hot water frequently applied to the abdomen often relieve the pain in it, which may render it necessary to use a cradle to keep off the weight of the bedclothes. The vomiting may be very troublesome, and you will find that very little reliance can be placed upon the drugs which are commonly recommended for it. If such of them as you choose to try do not speedily give relief, you had better wash the stomach out. In both the two last cases of peritonitis I have had under my care this has stopped the sickness.

Not much food should be given by the mouth; an ounce of milk every hour or half hour is quite enough, and if the patient is sick you must use enemata. Before beginning these, wash the rectum out with a few ounces of water at 100° F. From four to six rectal feeds in the twenty-four hours are enough; the first and third and fifth should be a nutrient enema. The egg-and-milk enema of our pharmacopœia is a good one, or you may take the yolks of six eggs, a drachm and a half of common salt, rather less than the same amount of pepsin, and seven fluid ounces of 0.15 per cent. solution of hydrochloric acid. Keep the mixture in a warm chamber for ten hours, and use four fluid ounces for each enema. The second, fourth and sixth feeds should be a nutrient suppository; those with a digestible basis are better than those having a basis of cocoa butter.

So much for the diagnosis and treatment of our case. We found at the post-mortem that she had acute general peritonitis, chiefly plastic, for there were only a few ounces of thin pus, but everywhere there was much thick lymph, especially in the region of the cæcum, where it formed a great

thick mass, hiding the cæcum, and making it difficult to find the appendix, so that at first sight we thought this organ had been the cause of the mischief, but it turned out to be quite healthy, and we found that there were several abscesses in the broad ligaments, from which the peritonitis had spread, in the first place, up to the cæcum, and that later it had become general. The structures in the broad ligaments were so distorted that it was very difficult to make their relationship, but there was every probability that the trouble began in one of the Fallopian tubes.

This seems a convenient opportunity for the discussion of the question of operation in cases of perityphlitis, or appendicitis, as it is often called. Three conditions may require operation—(a) acute general peritonitis, which we have already discussed. This occurs in less than 10 per cent. of all cases; (b) abscess, which also occurs in less than 10 per cent.; (c) relapsing cases.

If in any case of appendicitis the temperature remains up after the end of the fourth or fifth day, and the patient has not got general peritonitis, you should always suspect abscess, which is especially met with when there has been a very large amount of dulness and thickening in the cæcal region. The other signs are those which indicate an abscess elsewhere, such as a rapid pulse, furred tongue, irregular temperature, and rigors, together with, perhaps, local signs, such as a definite painful swelling, which becomes tender; but often from the general signs you may fairly conclude there is pus before the local have become marked, and you ought generally to make it out before redness, œdema of the skin, and definite fluctuation have appeared. The commonest seat for the abscess is just over the cæcum, but the pus not rarely travels upwards and backwards to the region of the kidney, and many cases of right-sided perirenal suppuration owe their origin to the vermiform appendix. I was able to show you an instance of this about a year ago. The pus may even travel up behind the peritoneum, and through the diaphragm into the pleura, where it sets up an empyæma. A case illustrating this was in the hospital under my care some time ago. Indeed, the appendix is so variable in length and direction, that it is a good general rule to remember this organ whenever you have to deal with any suppuration within a radius of six inches from the

cæcum. I have known an abscess starting in the appendix, to present between the liver and stomach, here the tip of the appendix lay close to the pancreas, also I have seen it present at Poupert's left ligament, and in another case it pointed through the right saphenous opening, and I have already referred to the instance in which it burst into the rectum. If you have made up your mind that there is an abscess, you must, if you can get at it, evacuate the pus and remove the appendix. This may be very difficult, and it may require most careful packing to prevent the pus from escaping into the peritoneal cavity. This happened in this case, in which the abscess presented between the liver and stomach, and also in that in which it appeared, near Poupert's ligament on the left. In each case the patient died.

The other condition for which operation may be required is relapsing appendicitis, in which it is often desirable to operate in between the attacks to prevent their recurrence. More than 80 per cent. of all cases neither develop general peritonitis nor abscess, and get over their attack without much difficulty. These cases are for the most part mild; there is probably some catarrh of the appendix, with a little local peritonitis around it, but this may be very slight. I have seen an appendix, which was quite thick and rigid from repeated attacks of catarrh, removed, and there was no appreciable local peritonitis. In a few cases there is an ulcer in the appendix, due to an impacted mass of hard fæces, and in excessively rare cases a foreign body lodges there and forms an ulcer. There is in the museum a grain of wheat which I found in an appendix. Taking all these 80 per cent. of the cases together, at least one in every four—I think the proportion is probably much higher—will have one or more other attacks, in each of which he may be one of the 10 per cent. who get acute general peritonitis, which is almost certainly fatal if suppurative, and probably fatal if plastic, or one of the 10 per cent. who get an abscess, and nearly half the cases of abscess die. Further, each attack is painful, and then compels the patient to stop away from his work—a matter of great importance if he is a wage-earner. Against these facts, you have to balance the fact that each attack gets less and less severe as a rule, and in each attack there is less chance of general peritonitis than in a previous attack, for in each attack

the local peritonitis shuts the appendix off more completely from the general peritoneal cavity. Then, too, the appendix is so variable in position that the surgeon may fail to find it. I have seen an instance of this quite recently, or it may be so bound down by adhesions, that it cannot be removed. Lastly, although the operation is now so perfected as to be one of the safest in surgery, yet no laparotomy is quite free from risk.

If your patient is an intelligent person, it will be your duty to put these facts before him, and to be guided by his age, his general condition, and his occupation; but you should usually advise operation if each successive attack is becoming more severe than the last; if each attack prevents the patient from following his occupation; if the previous attack was so severe as to endanger life, and if the interval between the attacks is getting shorter. If you do not operate, you will give opium in sufficient quantity to relieve pain and keep the bowels constipated. Leeches or hot fomentations may be applied to the cæcal region; small quantities of milk should be given at frequent intervals, and the patient should be kept in bed on his back until at least ten or fourteen days have elapsed since the temperature became normal and the pain disappeared.

Lastly, remember that there are few diseases about which more mistakes are made. I have several times known appendicitis said to be intestinal obstruction; twice I have seen it called typhoid fever. I have known it called perinephritic abscess, femoral hernia, muscular rheumatism, chronic constipation, pelvic cellulitis, ague, indigestion, and many other common things.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Reported by J. T. CONNER, M.D.

Senile Keratoses of the Hands and Feet.

A WOMAN, æt. 66, sent by Mr. Hitchens, showed considerable thickening of the epidermis of the hands and feet. On each sole was a yellowish,

translucent, horny plate, about an eighth of an inch in thickness, covering the heel. A similar plate covered the tread, whilst the hollow of the foot was unaffected. There were small horny plates on the toes, at the points where they pressed against the boots. The palms showed similar thickenings, but in less degree, and the fingers were also affected.

The disease had commenced twelve months ago in the feet, and six months later in the hands. Two years ago the patient received a severe mental shock, since which she had felt weak and low.

Mr. Hutchinson pointed out that the thickening of epidermis occurred only at those spots exposed to pressure. He accounted for the fact, that whilst symmetrical in the main, the disease passed farther to the ulnar side of the left hand, by differences in the manner of using the two hands in her house work. The predisposing cause was senility. Many old people had keratoses. They occurred in those whose nervous system was failing, and, as in the present case, might follow shock. He had known it to come on, in the soles of the feet, in an old man who was slightly paraplegic, after a fit of passion.

As the disease did not interfere with the patient, it was scarcely worth while to treat it. But if it was determined to do so, he recommended shaving the thickened epidermis, and then applying lanoline. Arsenic was of no service.

Purpura Hæmorrhagica in an Infant, following Eczema.

A female infant, æt. 5 months, sent by Mr. Hitchens, was the subject of this disease in a very peculiar form. The child was born at term, apparently healthy and well developed. When ten days old an eruption appeared on the head, which spread all over the body. It was very pruriginous, and evidently an attack of eczema of the ordinary form. It began to recede three weeks ago, and had now entirely disappeared from all parts, except the head and face. Two months ago epistaxis, lasting three days, occurred, since which the child lost flesh, and began to bleed from the mouth and ears, especially when she cried. Blood also oozed from the eczema spots, on the face and head, and from a small nævus on the back of the head. The child was suckled by the mother, who was in good

health, and whose diet was not of a nature to produce scurvy. There were five other children, all healthy, and no history of either syphilis or tubercle could be obtained. The eczema spots on the face and head now showed small blood crusts, each about the size of a sixpence. The skin elsewhere was normal, except for the occurrence of a few bruises on the trunk and limbs. The largest of these, about the size of a crown, was on the abdomen.

The skin in the neighbourhood of the anus was eczematous. It was red and glazed. Mr. Hutchinson pointed out that the appearances here exactly resembled those of syphilis. He did not suggest that diagnosis, but, on the contrary, adduced the fact as an example of the fallaciousness of basing a diagnosis of syphilis, merely on the appearance of an eruption. No eczematous or other eruption was to be seen on any other parts. But some bruises, in the form of greenish-yellow stains, were present on the trunk and limbs. The largest, about the size of a crown, was on the abdomen. Mr. Hutchinson considered the case one of purpura hæmorrhagica. But it was remarkable that no hæmorrhages into the skin were to be found anywhere.

Ulcerating Nævi on the Buttocks of Two Infants.

These cases, brought by Mr. G. W. Sequeira, were exactly similar. The children were of the age of five and three months. Both showed a superficial (capillary) nævus near the anus, about the size of a crown, which was ulcerating in the centre. Strange to say, both had also a nævus on the forehead, which was not undergoing any destructive changes. The elder child was produced on a second occasion, when the nævus on the buttocks appeared to be spreading at the edge while healing in the centre. It was pointed out that spreading and ulceration did sometimes occur in nævi, and that the ulceration in the present cases was probably due to irritation from the urine and fæces.

Lupus Erythematosus, with Unusual Condition of Scar.

A man, æt. 36, sent by Mr. Hitchens, had this disease on his nose and cheeks in the form of usual "butterfly" pattern. The erythema was in

patches, alternating with scar. The remarkable feature was that the scar was of unusual thickness, and looked like the ivory patches of morphœa. In front of the ears the scars were deeply pitted like those of scrofula. The ear lobules were destroyed. On the scalp was a patch of scar about the size of a shilling, bald, and also deeply pitted. The patient gave the following history:—From the age of 10 to 15, he suffered from ulcerating chilblains. From 15 to 18, he had large scrofulous glands in the neck, with suppuration on the left side. When he was 21, the ears became sore in winter, and wept. At 26, a red patch, about the size of a shilling, appeared on the left cheek. This remained quite stationary for four years. At 29, three patches appeared, at the root of the nose and on either temple. From these the disease spread until it assumed its present condition. The patient's sister had died of consumption, and he was, in youth, suspected to have that disease. This association was pointed out to be much more usual in lupus erythematosus than in vulgaris. The diseases were contrasted in other respects. The former might infect distant parts, but tended to wear itself out and get well in time; whilst the latter never infected distant parts, except at its very beginning, and had no tendency to spontaneous cure.

Raynaud's Disease, accompanied by Severe Hæmorrhages.

The patient was a Jewess, aged 25, brought by Dr. Teuchmann. She had been subject to attacks of blueness and coldness of the hands and feet since ten years ago, and, when a child, had suffered from chilblains. Two years ago she had severe epistaxis. This had occurred again during the last three weeks, accompanied by bleeding from the gums, and hæmaturia.

The fingers were cold and dusky, the nails were thinned, and had lost their convexity. All over the hand were scaly patches. There was no scleriosis of the skin, nor was there gangrene of the tips of the fingers, but simply peeling of epidermis. The nose was in a similar condition to the hands—blue, dusky, and shiny. On the cheeks were a number of scattered erythematous papules, and the ears were shrivelled.

Though no history of gout in the family could be obtained, Mr. Hutchinson considered that the

hæmorrhages were the result of the inherited form, remarking that Jews were frequently gouty, as well as bleeders. At the same time it was to be remembered that it was not uncommon to have the urine bloodstained during the paroxysms of Raynaud's malady (paroxysmal hæmatinuria). In this case, however, as regards the gums and nose, it was free hæmorrhage. The case was, he remarked, a typical one of the recurring form of Raynaud's malady. Yet there was no actual gangrene. It was a condition to which Raynaud's term "asphyxia of the extremities" was applicable.

POSTSCRIPT.—A fortnight later, Dr. Teuchmann reported that this young woman had developed bronchitic symptoms, and had died. It had not been possible to obtain an autopsy.

CLINICAL LECTURE

ON DISSEMINATED SCLEROSIS.

BY
JOHN ROSE BRADFORD, M.D., F.R.S.

GENTLEMEN,—The reasons for giving a lecture on disseminated sclerosis are—first, it is one of the commonest of the spinal scleroses; second, it is the sclerosis that is least often diagnosed, and you can further say that it is the most important to diagnose; and, thirdly, it runs a fairly acute course for a sclerosis, rarely lasting more than ten years, sometimes as short a period as two years, the average being six years.

For the reasons, then, that it is so common, that it is not easy to diagnose, and that it is fairly acute in its course, it is worthy of attention. Further, it is more important to us than all the other scleroses, because it is so protean in its manifestations. Moreover, owing to the great mistakes that are made in practice, in diagnosing this complaint, it must receive very careful attention from us.

It is a disease which is usually said to be equally common in both sexes, but the modern views are that this is not quite true; in reality, it is more common in women than in men—that is, if

you are including the abnormal types, not the classical type described by Charcot. The usual text-book statement of course is, that there is no great difference in the sexual incidence.

The etiology of disseminated sclerosis is at present obscure; there is not much information on the point, less than what is known about most of the other scleroses. One definite thing is, that it commonly occurs from twenty to thirty-five years of age; it is rare over thirty-five, and it is doubtful if you ever see it in young children. There is a possibility of error in diagnosis between disseminated myelitis and disseminated sclerosis, and it is to be noted that the former disease occurs not infrequently in children. As regards other influencing causes, one of the most striking, at any rate in the atypical forms, is depressing circumstances generally. A large number of cases of disseminated sclerosis occur in young women after disappointments in love matters, and so forth, and various home discomforts. There is no doubt that one is much struck with the great frequency of slight cases of this disease, following influences of this kind. Other causes are, excesses of various kinds; such as over-work and over-anxiety; and another thing which is held to be the cause is heredity, *i.e.*, descent from a neurotic stock; however, it is uncommon for patients with disseminated sclerosis to have parents suffering from that disease, but they may have allied nervous disorders. Two other factors to be considered are traumatism and acute specifics. As regards traumatism, there can be little doubt but that disseminated sclerosis does follow injury; thus, a girl developed disseminated sclerosis after scalding her arm with hot water. If you have a history of nervous symptoms after traumatism, you should not be too hasty in ascribing them to hysteria. Convalescence from acute specifics is another cause; but the acute specific that is held to be most important is typhoid fever. Syphilis is probably very unimportant. You can therefore see that the whole question of the etiology of this disease is summed up by saying that it is obscure, with the exception that it has a definite age incidence, and syphilis has practically nothing to do with it. I emphasise the latter point, owing to the fact that syphilitic disease, particularly in the cerebral meninges, is liable to produce symptoms resembling disseminated sclerosis.

As regards the kind of disease that disseminated sclerosis is, it is held to be a random sclerosis. The teaching is that there are at least four kinds of sclerosis—

1. Consecutive sclerosis, which are really degeneration of certain tracts following some other lesion of the nervous system, as, for example, the lateral sclerosis that follows hemiplegia or transverse myelitis.

2. System sclerosis, such as tabes.

3. Combined sclerosis, such as Friedreich's disease; and

4. Random sclerosis, of which disseminated sclerosis is supposed to be the instance. You understand by random sclerosis, a random distribution of the masses of fibrous tissue, sometimes in the white matter, sometimes in the grey matter. There are no two cases of disseminated sclerosis that exhibit precisely the same lesions post mortem. It is a disease which is extremely variable as regards the disposition of the lesions.

The probable pathology of the disease is contained in the supposition that it is a disease which perhaps begins round the blood vessels. Formerly it was taught that all sclerosis started from blood vessels, but now such system sclerosis as tabes are held to be due to a primary atrophy of the nerve fibres. Disseminated sclerosis is still held by many to be a vascular sclerosis, since, in the islands of fibrous tissue it is not uncommon to find a blood vessel, and a further reason for this view is that pointed out by Charcot, *i.e.*, the nerve fibres can be traced through the masses of sclerosis, and since the nerve fibres are not destroyed primarily. It is only quite late in the disease that you get the destruction of the nerve fibres, and there is then ascending and descending degeneration. On the other hand, the only reason for thinking that the disease begins in the nerve elements is the wonderful clinical resemblance between different cases. The clinical resemblance is greater than the pathological resemblance, the great bulk of cases of disseminated sclerosis resemble one another clinically at some period or another in their course. If you admit that, it is difficult to suppose the disease is absolutely random in its distribution.

Disseminated sclerosis is best described by recounting the varieties. Taken as a whole, the disease is difficult to describe. You can give an

account of how the disease begins in a given case, or you can set out the whole clinical picture of the different forms of the malady.

It is most convenient to speak of a spinal type and a cerebral type, but they are not usually seen independently. The disease is usually of the cerebro-spinal type, with the predominance of one or the other form. Some recognize three types—spinal, bulbar, and cerebral. The atypical forms of disseminated sclerosis are usually of the spinal type; the typical are usually of the cerebral type. I shall begin with describing the spinal type.

With the spinal type the patient has certain motor symptoms which we will now go into. The first motor symptom is usually weakness, *i.e.*, palsy. It usually shows itself, first of all, in the patient's gait; the patient has paresis rather than paralysis, and the gait of disseminated sclerosis is described as one of two types. You may have either—the spasmodic type, in which the patient has difficulty in walking, from stiffness in the legs and the toe catching in the ground; they may even be actually thrown to the ground; they may tell you that they sometimes fall; the other type of gait is the so-called cerebellar gait. They reel to and fro; they stagger, and are unable to maintain the erect posture. The legs are wide apart, and they stand with as wide a base as possible. It must be remembered that you may have the paresis in only one leg, but that is not very common; but you may say that it often begins with weakness and rigidity of one leg. Then occasionally the weakness begins first in the arms, and then when it does that the patient usually presents himself for observation, owing to some failure in writing, suggesting, as I have seen, writer's cramp, or difficulty in sewing, and so forth. When it begins like this in the arms, it practically always affects one arm only at first. According to some, it would be more accurate to put these arm cases under the head of the cerebral type. These are the motor symptoms in the spinal type. In the spasmodic type the reflexes are, of course, increased, and you have well-marked ankle-clonus: it is not the increased knee-jerk which is so important, but rather it is the presence of ankle-clonus. In the type where the patient has a "cerebellar" gait, it is not uncommon for the knee-jerk to be lost; it is not necessarily lost, but you may have the knee-jerk absolutely lost, and,

under these circumstances, there may be some difficulty in the diagnosis at first.

As regards the sensory symptoms in the spinal type, they are not well marked ; but these patients with marked spinal symptoms not uncommonly have hemianæsthesia, which is usually perfectly limited to the middle line, and involves all forms of sensation. One thing, perhaps, you might say about this hemianæsthesia is, that it is liable to be present one day and absent on another, and that is another point which may put you off your guard.

The next set of symptoms in the spinal type are the visceral symptoms. In all these cases they get trouble in the viscera, it usually shows itself by incontinence of urine, but this is usually transitory, the visceral functions again becoming normal. The early and transitory incontinence of urine and the early and transitory retention of urine are among the signs of the disease which will enable you to diagnose it. In advanced cases of the spinal type, and by spinal type you do not mean that the only lesion is in the cord, sometimes the symptoms are least marked in the spinal system ; in advanced spinal cases I say you may have trophic lesions, bedsores, affections of the nails, and glossy skin, etc. : these do not form marked features of this disease.

As regards the symptoms in the cerebral type. Cases of disseminated sclerosis have sometimes a certain disturbance of the mind, it shows itself by progressive involvement of the intellect with loss of memory, and so on, and there is one curious and characteristic fact to be noticed, the mental condition does not coincide with the physical condition, they are often very debilitated, but they are mentally quite happy, and very pleased with themselves. You may see a patient unable to stand or walk or sit up, who breaks glasses and cups with his tremor, and you may see such a patient smiling and laughing all day long, as I say, quite pleased with himself. That is a characteristic feature of the disease, and it is but rarely that they are melancholy. They are, to a certain extent, emotional, but they laugh more than they cry. These mental symptoms occur late in the course of the disease. One symptom described under the cerebral type is the tremor. This tremor is very characteristic ; there are two adjectives which characterise it. First of all, it is

an "intention" tremor : it is absent during sleep, and comes on with exertion. But a very much more characteristic thing is the massiveness of the tremor, it involves the whole limb ; you never see a case of disseminated sclerosis with tremor only of the hand or of the fingers, the tremor is in the whole arm, involving the muscles of the shoulder as much as it does the muscles of the wrist, and it differs from the other tremors known in medicine in several points. It is different from the tremor of paralysis agitans, which affects particularly the fingers, there is not much difficulty in this diagnosis, because you will not see a case of paralysis agitans with massive tremor. The tremor involves the head, that is to say, the head may shake on its own account. In paralysis agitans the head shakes because the body shakes. In disseminated sclerosis there is no tremor when the patient lies down, but on sitting up the tremor is so violent that the jaw may be fractured by contact with the chest. The tremor may ultimately involve the entire body. The tremor may be an early or a late symptom : in the cerebral cases it is usually an early symptom ; in the spinal cases it is usually a late symptom, you must not, therefore, lay too much stress in the early diagnosis of this disease on the absence of tremor.

The next cerebral symptom is, of course, the speech ; and I do not think it is much use to describe the speech of disseminated sclerosis, you have to listen to it to understand any description, it resembles the speech of the patient with general paralysis of the insane ; but there is not quite so much slurring, and there is perhaps a tendency to separate the syllables, giving the speech its staccato character. It is difficult to give any single description which will apply to all cases, because they vary so much, in advanced cases the difficulty in speech rapidly increases until the patient becomes speechless as in paralysis agitans, and you may see a patient with disseminated sclerosis who understands everything that is said, but yet is unable to say anything to the bystanders. This speech difficulty is sometimes described as a bulbar symptom.

There is another important group of symptoms in relation to the eye-muscles. Most cases of disseminated sclerosis have an external and partial ophthalmoplegia at some time or other, in other words diplopia is a symptom of this disease.

Hemiplegia occurs not uncommonly, and this hemiplegia is then of sudden onset, and there is nothing to distinguish it from any other hemiplegia except the fact that it is liable to be transitory; it is a very interesting fact, and must be remembered that these hemiplegic symptoms are liable to be transitory, for it is very misleading to meet with extensive palsy coming on suddenly, and as suddenly disappearing.

Other occasional symptoms you may meet with are attacks epileptiform and apoplectiform in nature. The apoplectic forms are shown in attacks of sudden unconsciousness, the patient falling down and remaining unconscious for several hours, gradually recovering, and when he has recovered he may or may not be hemiplegic, but he will not suffer from cerebral hæmorrhage; hence, it is an instance of true apoplexy, a condition in which a person becomes suddenly unconscious, but where there is no gross lesion post-mortem to account for the trouble, and sometimes patients with disseminated sclerosis die from similar attacks to those seen in patients with general paralysis of the insane.

Epileptiform attacks are also not uncommon. There was a man in Dr. Poore's ward who had forty fits in one day, and it is difficult to distinguish them from ordinary epileptic attacks.

In regard to the diplopia mentioned among the eye symptoms, it is a characteristic thing, and it usually depends on the weakness of the external rectus muscle, and it is further characterised by a tendency to be transitory. You ought always to be very careful to ask for a history of diplopia; it is an early symptom, and may be transitory. It is equally characteristic to find no internal ophthalmoplegia. The pupil in disseminated sclerosis is very rarely affected; some authorities say it is unequal, but it reacts to light and accommodation. Great stress should be laid on the fact that the pupil always reacts to light and accommodation. There is no loss in the reflex of the pupil. These are the principal symptoms referable to the external muscles of the eye-ball. In regard to the retina and the optic nerve, 60 per cent. of the cases of disseminated sclerosis have some visual trouble, and these are very various, sometimes a central scotoma, sometimes a central blurring or obscuration of the field of vision. A more characteristic symptom is, that the field of vision is

smaller than it ought to be, either in one or both eyes, and this diminution in the field of vision is an important matter. Does the restriction of the field of vision involve all or only certain of the colours; generally you have restriction of the field for white light, and I am laying stress on this point, because it is by this means that it is asserted that you diagnose hysteria from disseminated sclerosis. The white field of the normal retina is bigger than the coloured field, the delicate tinted fields are the smallest. It is held by Dr. Buzzard that in the restriction of the field of vision in disseminated sclerosis the normal retina is, so to speak, simply smaller, and you do not get achromotopsia alone, in which the field is diminished to coloured light only. It used to be held by Charcot that a patient who had great restriction in the field of vision for coloured objects, but little or none for white light, was hysterical. On the other hand, if the visual field is restricted for coloured light and for white light, it is probable that there is organic mischief.

It is difficult to say what is the cause of this restriction of the field of vision, but the typical disturbance is a peripheral limitation of the field of vision involving all the colours. Whether you are justified in saying that a particular case is functional because the loss is to colour and not to light, is perhaps doubtful. Those are the principal results, as far as relates to what the patient himself complains of; on examination of the fundus there are changes in the optic nerve in perhaps more than half the cases, and the usual thing is of course varying degrees of optic atrophy, from a slight atrophy to complete atrophy, with amaurosis. What is more important, indeed, is the fact that in disseminated sclerosis you may have slight optic neuritis. I do not think you get very intense optic neuritis in this disease.

The principal points, then, to note are the diplopia, the absence of any iridoplegia, the presence of the restriction of the field of vision and the presence of changes in the optic papilla, the palsy, the occasional hemianæsthesia, the characteristic gait, the tremor, cerebral symptoms, and what I have left to the last—nystagmus. You probably never get disseminated sclerosis without nystagmus, but of course the converse is not true: and where you suspect so-called functional derangement, you should avoid being dogmatic. Before

deciding on the nystagmus, you must make quite certain that there is no error of refraction, and that the patient has not followed any occupation in a bad light. The nystagmus in disseminated sclerosis is a horizontal nystagmus, and it is usually equal in both directions, not being more marked to the right than to the left. It is met with in two forms; sometimes it is always present, and at other times, when it is slight, the patient must look particularly at an object in order for this sign to be detected. The first form is more important in the diagnosis than the detection of slight nystagmus brought out on extreme movement to the right or to the left.

In reference to the diagnosis of disseminated sclerosis, it may be very easy, and it may be extremely difficult. If you have tremor, the speech difficulty, and nystagmus, it is easy, but the tremor and the speech are late symptoms, and you may have considerable difficulty in diagnosis in the spinal type, in which form you are probably justified in suspecting disseminated sclerosis if the patient has weakness of the leg, trouble in the gait, increased knee-jerks with ankle-clonus, with nystagmus, provided, of course, they exhibit no further symptoms indicative of some other gross lesion. Thus, of course, cerebral tumour will give these symptoms, but then there would be also vomiting and headache. The diagnosis may turn on the nystagmus, there are many diseases which produce weakness of the legs and increased knee-jerks, such as caries of the spine and transverse myelitis, but they do not produce nystagmus. To assist you in the diagnosis you must inquire for these two symptoms, *i.e.*, the transitory diplopia, and the transitory incontinence of urine, patients with chronic disease of the spinal cord do not exhibit transitory affections of their sphincters, they do not have transitory recurrent attacks, and it is the same in the case of the diplopia. Whatever you do be very careful not to exclude disseminated sclerosis, and diagnose hysteria because of the transitory condition of the palsies.

The principal diseases to diagnose disseminated sclerosis from are, first, hysteria, or so-called functional disease; secondly, cerebral tumour; and, thirdly, general paralysis of the insane. There are, of course, other maladies, such as paralysis agitans and Friedreich's disease, but the diagnosis from them is, so to speak, a matter of

scientific interest; but it is a question of great practical importance in regard to diagnosing this disease from hysteria, it is impossible to warn you too much against loosely diagnosing functional disease and hysteria, the mistake is nearly always made owing to the stress laid upon the transitory character of the lesions. How, then, is it to be distinguished from hysteria? The first thing is that disseminated sclerosis may be preceded by a period in which the patient only exhibits functional symptoms. Disseminated sclerosis is held to be preceded by functional disease, and you must bear in mind that these so-called functional paralyses in young people may apparently go on to disseminated sclerosis, some, as you know, even hold that there is no such thing as functional disease, either they are malingering or they have a definite organic disease. Great harm may be done to the patient and to yourself by the hasty diagnosis of functional disease. The first thing is to note that nystagmus and the diplopia and the history of any sphincter trouble, the presence of any visual disturbance, and there is one physical sign which you must note, and which I have left out till now, and that is the plantar reflex. It is held that in hysterical palsies the plantar reflex is lost, and it is not lost in disseminated sclerosis. Difficulty arises in the case of a young man unable to walk, with doubtful nystagmus, with doubtful ankle-clonus, and with doubt, perhaps, as to the condition of the plantar reflex; but stress should be particularly laid on the history of diplopia and sphincter trouble, if you have either or both of these you have no right to diagnose hysteria or functional trouble, for though the patient may have hysteria in addition, that does not disprove disseminated sclerosis, it is a disease which comes on under those circumstances that produce hysterical symptoms.

The next disease to diagnose disseminated sclerosis from is tumour of the brain, and the most careful observers may go wrong in this point, for now and then you see a case of disseminated sclerosis diagnosed as cerebral tumour, and sometimes you see the converse. Thus, for instance, disseminated sclerosis has been diagnosed, but, in reality, it is a case of cerebral tumour; there are tumours in certain positions which will produce weakness of the legs, ankle-clonus, tremor, and

nystagmus, so as to simulate disseminated sclerosis most closely, and the most common place for these tumours is in the pons or in the white matter of the cerebral cortex. The distinction is sometimes to be made entirely on the presence of optic neuritis, and that is why I warn you that if there is intense optic neuritis the probability is that it is a case of tumour. It must also be remembered that a tumour in the cerebellum is liable to produce optic atrophy and also diplopia, and, as I said at the beginning of my remarks, that cases of disseminated sclerosis may exhibit loss of knee-jerk and ataxia, and hence cause a difficulty in diagnosis. You have to rely mainly on the headache and vomiting. Cerebral tumours in the white matter may produce a tremor which is indistinguishable from the tremor of disseminated sclerosis, a subcortical tumour may do this.

To make a mistake between general paralysis of the insane and disseminated sclerosis may be followed by awkward consequences, for the patient with disseminated sclerosis is not liable to become extravagant. The mistake is easily made from the tremor and the speech. The speech defect is very similar in the two diseases, and tremor occurs in both; in general paralysis of the insane you have inequality of the pupil, and you get definite sluggishness or loss of movement, which you do not observe in disseminated sclerosis.

As regards paralysis agitans there is a difference in the character of the tremor, and there is also the marked difference in the age incidence, it is uncommon to have paralysis agitans in the young, and when it does occur in the young it runs a very acute course; of course, the principal points of difference are the spasmodic rigidity, the fixed expression, and the "poker back." These are the principal points in which you make the mistake in the diagnosis between the two diseases, if you rely on the tremor for disseminated sclerosis you may fall into error, but if you rely on the fixed expression, and the eye symptoms for paralysis agitans, you will avoid making a mistake.

In Friedreich's disease or hereditary ataxy, you have nystagmus and ataxic gait, and speech defect with loss of knee-jerk, so that it somewhat resembles certain forms of disseminated sclerosis, but the two most important distinguishing points are spinal curvature in Friedreich's and the Z-shaped foot.

The prognosis in disseminated sclerosis is never satisfactory, and of that I suppose there is no doubt; but like other nervous diseases it may undergo arrest, and it is asserted that it may undergo amelioration, understanding by that the disappearance of the hemiplegia or the fits, or anæsthesia, but one thing is quite certain, the patient never gets well again, if the patient does recover your diagnosis has probably been wrong. With regard to the patient's death, six years is the average duration of the disease, it may be as short as two or as long as fifteen. These patients die from various causes, first from choking as in general paralysis of the insane, their bulbar symptoms may be the reason of their choking, just as occurs in patients with progressive muscular atrophy. More usually death arises from cystitis, bedsores, and complications of that description, occasionally they succumb to tubercular lung lesions, or they may die from simple lung complications, low forms of pneumonia, such as carry off paralysed patients. Sudden asphyxia is also a danger. Occasionally these patients die in the epileptiform and apoplectiform seizures.

As to treatment, I am afraid there is no satisfactory treatment, there is no known nervous treatment directly applicable to this complaint. Complications may be warded off by suitable nursing, and some improvement in general health obtained by the use of arsenic, and it is usually prescribed with strychnine and belladonna.

REVIEW.

Kelly's London Medical Directory.

This is the eighth year of the appearance of Messrs. Kelly's work, and we certainly think by now they might have established such communications with societies, publishers, etc., as would enable them to issue the volume with information corrected nearly up to date. We notice a medical man, deceased in January, 1894, still editing a weekly journal, and the officers of a society holding on from 1894 to 1896 without alteration. Such inaccuracies seriously detract from the value of the work, which otherwise contains much valuable information.

THE CLINICAL JOURNAL.

WEDNESDAY, MARCH 25, 1896.

A POST-GRADUATE LECTURE

ON

ALCOHOLIC INSANITY,

Delivered at the Bethlem Royal Hospital, March 3rd. 1896,

By MAURICE CRAIG,

M.A., M.B., B.C., M.R.C.S., L.R.C.P.,

Assistant Medical Officer to the Hospital.

GENTLEMEN,—In introducing my subject, I must point out that alcohol is the most common general cause of insanity in this country. In 1859, Lord Shaftesbury, before a Select Committee on Lunatics, expressed his opinion that about 50 per cent. of the cases admitted into English asylums were due to drink, and quoted Esquirol in support of that statement. Later statistics, however, show that from 10 to 20 per cent. is nearer the truth; at this hospital about 12 per cent. is the average. Nevertheless, even our present figures show what an important part is played by alcohol in the production of insanity. In compiling statistics, great care must be taken not to render them fallacious. Friends often tell us that the cause of the patient's break-down has been drink, preferring to give this cause rather than to acknowledge the fact of an insane heredity in the family; but I need hardly point out how necessary it is to inquire carefully as to the duration of the excessive drinking habits. Sometimes, we find that has only lasted a week or two, or at most a month; and we must not forget that giving way to vicious habits may be an early *symptom* of mental disease, not really the cause. Moreover, we have seen that intemperance may be one of the moral perversions not uncommonly left by a former attack of insanity.

The effect of alcoholism on parents is not only manifested by impairment of their own mental faculties, but in their offspring it engenders a tendency to intemperance, epilepsy, or idiocy, as well as to insanity.

Almost every form of insanity may be started by

alcohol. For convenience of illustration I have adopted the following classification :—

1. Drunkenness.
2. Delirium tremens.
3. *Mania a potu*.
4. Chronic alcoholism.
5. Dipsomania.

Drunkenness is usually induced by a large quantity of alcohol being taken within a short space of time, and the mental aberration thus produced is, as a rule, temporary. But in certain predisposed persons it may be the determining factor of acute delirious mania. It must always be borne in mind that even small amounts of stimulant may suffice to intoxicate epileptics, those who have been the subjects of sunstroke, or who have sustained head injury. One may frequently see cases in whom one glass of such intoxicant will upset the mental equilibrium. Alcohol exaggerates a person's *normal* temperament, and this may be seen in every form. Thus, a normally weak-minded person becomes, under its influence, foolish; unusually morose individuals weep, while a man who is normally excitable is rendered by alcohol merry, or even witty. Dr. Wilks said that alcohol lets one see into the innermost nature of an individual, and that in order to witness the earliest symptoms of general paralysis of the insane, one has only to go to a large football dinner.

Delirium tremens is brought about by taking large quantities of alcohol within a comparatively short time. This class of case is not often met with in asylums, as the attack is usually of short duration. Still, there are a few points of interest to which I might draw your attention.

During the attack the patient is undoubtedly not of sound mind, and we must remember that anything which gives rise to temporary insanity may lead to a more permanent derangement. In other words, certain individuals, especially those with a neurotic inheritance, may not recover from an attack of delirium tremens in the usual way, but the symptoms may pass into those of acute mania, or acute delirious mania, or stupor, or indeed any other form of insanity.

This is the case not only with alcohol, but with other drugs. For example, it is not rare to hear of patients being put under the influence of chloroform, and instead of recovering from its effect, they remain in a stuporous condition, or may become acutely excited. We had such a case here last year, that of G. W., æt. 17, who was admitted here on March 21st, 1895. His occupation was that of paper-maker. This was his first attack, he had been ill twenty-six days, and was said to have always been sober. The family history was good; his mother had had paralysis agitans, and one sister had died of phthisis, and that, apparently, was all the history that could be obtained. On February 22nd, he was given ether, in order that a tuberculous sinus connected with the os calcis might be scraped out. He did not come round from the anæsthetic, but remained in a confused condition, and on February 25th he began to have hallucinations of sight. On the night of the 26th, he was very excitable, and on the 27th, maniacal. Before admission here he had, for ten days, been very talkative and incoherent, but the night previous to coming in he had relapsed into a depressed condition. He had also passed everything under him for the preceding fortnight.

On admission he was quiet, and refused to converse, but observed what was happening around him, and took food himself. His optic discs were normal, the tongue furred, bowels constipated, and the pupils were equal, with normal reaction to light and accommodation. There was disease of the right knee-joint, which was put in a Croft splint. He used to complain of seeing eagles flying about the ward, and also stated that he felt as if he were going up in a balloon from time to time. Sometimes he accused us of poisoning his food, but only on a few occasions. He remained here until April 20th of last year, by which time he had much improved mentally, and would converse rationally. Hallucinations still persisted, but became less marked. His knee-joint, however, was getting decidedly worse, he began to get pyrexia of an hectic type. A consultation was held, and it was decided it was necessary to amputate the limb. But here arose again the question of an anæsthetic, and that leads me to remark that the fact of a patient failing to come round under its influence once, does not contraindicate its future use in the same individual. We, accordingly, granted our patient leave of

absence for a month, in order to have the leg removed, and the operation was done soon after he left. The anæsthetic seemed to do him no harm, he had no return of his mental troubles, but made steady improvement, and was discharged well on June 3rd. We have a lady inmate who was put under an anæsthetic for opening an ischio-rectal abscess, after which she became acutely maniacal. As a point of interest, I would call your attention to the fact that after confinements, in which it has been necessary to administer anæsthetic such as chloroform, a woman may have an attack of insanity, but at succeeding labours its administration may have no after-effects.

To return to delirium tremens, the onset of that condition is not nearly so sudden as is often supposed; it is usually preceded by a period of increased nervo-muscular excitability. The person is impulsive, gets into a sudden passion without adequate cause; becomes afraid of things, suspicious, restless, and gloomy. These troubles become increased towards night, the patient does not sleep, and misinterprets every sound which reaches him. He also develops somatic symptoms, gastritis, tremor, and so forth. As time goes on, hallucinations begin to appear, first at night, and are most commonly visual; their character constantly changes, and the patient is terrified by them. In this they differ from the hallucinations of acute mania. An alcoholic patient is afraid, and vainly endeavours to get away from his hallucinations, while, on the other hand, an acute maniac shows no alarm.

As the alcoholic case progresses, the hallucinations appear during the day as well as at night. During recovery these hallucinations disappear in the inverse order. Probably they are manifested at night, first, because patients are quieter and are left to their own thoughts. Delirium tremens has to be diagnosed from acute delirious mania. In the latter the temperature is raised, the hallucinations, though abundant, are not terrifying, and, in addition, in delirium tremens we are guided by the somatic symptoms and by the history. There is one form of delirium tremens in which the temperature is high, but in the ordinary kind the temperature is not much above the normal. Of course, the history of the case may not assist you, as it may have been an ordinary alcoholic case, and have passed on to delirious mania.

To sum up, the risks in delirium tremens are five in number.

1. The patient may pass into a condition of stupor, coma, and death. One frequently sees such a termination outside.

2. The hallucinations may persist after the other symptoms have subsided.

3. Delusions of persecution, poisoning, etc., may persist.

4. After an attack of delirium tremens the finer attributes of the patient's character may become dulled, and he may, in consequence, manifest lazy and immoral habits. This is frequently met with in those who have had an acute alcoholic attack. They have, probably, been most assiduous in business matters, hard-working and punctual, but after apparent recovery, are lazy and have a tendency to immoral ways.

5. The last risk is, that the case may pass into acute mania, but such cases are generally pre-disposed to it.

Occasionally, during an ordinary attack of delirium tremens, we see a patient suddenly become exalted with the idea that he is an emperor or God, his former fear and misery having left him. This, probably, will only last a short time; it may be half an hour, an hour, or a few hours, and he will then relapse into his former restless and terrified condition. This is known as the *ambitious* form of delirium tremens, and is not common, being usually confined to persons with insane inheritance. It nearly always indicates intellectual ruin, and the prognosis is grave.

There is another form of acute alcoholism which, by some, is erroneously classified as synonymous with delirium tremens, namely, *mania a potu*. This is a very important form of insanity, because the subjects of it often give a lot of trouble. There is not the same degree of physical prostration in this as in delirium tremens. Sufferers from delirium tremens look ill, but subjects of *mania a potu* appear in their usual health, or at any rate their physical condition does not appear to be markedly affected. Usually, as soon as the alcohol is withdrawn and the patient is placed under definite *régime*, he rapidly gets well. These patients frequently have an insane inheritance, and very little drink will produce the condition; it is met with in epileptics and in degenerates generally. They suddenly become violent, and may be

homicidal. In a private house it is most difficult to treat these cases, and yet experience teaches us that if vigorously attended to they soon recover. When transferred to an institution their recovery is sometimes so rapid that the friends and the patient do not recognize that detention was necessary. They are then apt to turn on the medical attendant, and say that the step ought not to have been taken, and may even go so far as to institute legal proceedings. Such cases are very awkward, because their presence in a private house is a source of danger; they may attempt to kill themselves or those around them. Besides, in a private house it is not at all easy to keep the patient away from alcoholic liquor, whereas in an institution no such risk is run. I may remark that alcoholics are distinctly a litigious class, and practitioners are consequently sometimes afraid to sign a certificate for such. But there is very little risk if the practitioner calls in other opinion before the step is taken. I will read you an account of a case of *mania a potu* which we had here last year.

W. M. is a man we have known for some years. He was admitted, in the first place, on September 22nd, 1890, profession, commercial traveller, æt. 34, two children, youngest 18 months. He had then been, more or less, insane for a period of nine months; the cause given being intemperance. He was suicidal and otherwise dangerous. Family history: his father's brother was insane, and a cousin also. The patient had had syphilis two years before admission, and had always been inclined to drink intemperately. During the month before coming in he became extravagant and erratic. He began by buying horses which he could not afford to keep, and stayed out all night. A fortnight prior to admission he commenced to be violent and suspicious. He fancied he was worth a million of money, thought his wife put poison in his food, and that his friends wished to kill him. He was also hostile to his wife, and threatened to injure her and others of his family. The night before admission he passed the night in a state of nudity in a field.

On physical examination, his tongue was found to be furred, and had a slight tremor; urine normal, pupils equal, but sluggish to light. The knee-jerks were diminished. He was very talkative, and boasted of his strength, and of his skill in riding and shooting. I notice that he was diagnosed

on this occasion as a probable general paralytic, but it subsequently became evident that was not the case. On September 29th he, for the first time, denied that he ever threatened his wife, and at this date is noted to be happy and contented in the hospital. On October 4th, he said his wife had been false to him, and hinted at adultery. He improved, and on October 28th was sent to our convalescent home at Witley, where he remained for a month; afterwards he was allowed away from the hospital on leave of absence for a month, and was discharged well on December 10th, 1890. We do not appear to have heard anything more of him until December 15th, 1894. The history then was that he had not drunk much alcoholic liquor since his discharge, until a few months before the date I have just mentioned, when he again became extravagant, and bought things for which he did not pay, did not discharge his cab fare, went with disorderly women, ill-treated his wife, and had not been near his business for weeks. This time he was found sleeping on the road-side, and he had threatened to commit suicide. On admission, his general health was good. On the day following he appeared rational, said he was quite willing to remain in the hospital, and that it was right for him to be here. By January 10th, (not a month after admission) he was quite friendly to his wife, and after two months leave of absence, he was discharged well.

On November 5th, 1895, he was re-admitted into the hospital, with a history of drinking to excess. He had been more or less depressed since the early summer of 1895. He had become very excited, walked about half-dressed, bought horses and carriages which he had no prospect of paying for, and again accused his wife of unfaithfulness, (we were able to prove that there was no foundation for that), and threatened to kill her. His general health was good, and nothing abnormal, except tremor of hands was noticed. He was boastful, and practically showed a repetition of his former symptoms. By November 7th (two days after admission), he was rational in conversation, but was still hostile towards his wife. He made steady progress towards recovery. On November 17th, he still made unfounded accusations against his wife, but from his previous attacks we had noticed that that was the last symptom to disappear in the process of recovery.

As he had not been *seen* by a magistrate before admission "the notice of right to a personal interview with a Justice" was given as required by the Lunacy Laws. This he signed, and in due course was visited by a Justice of the Peace. Except for the hostility to his wife (which we had always observed in his case was the last symptom to disappear) he had nearly recovered; and it is not to be surprised at that the magistrate, though against our expressed opinion, notified in his report to the Commissioners in Lunacy that the patient had now recovered. A few days later we received a communication from the Commissioners saying that unless fresh symptoms of insanity had developed, the patient must be forthwith discharged. There was no option but to discharge the patient, though we were fully cognisant of the possible consequences of such a step. He asked to be allowed to remain as a voluntary boarder, and go to our convalescent hospital in the country. He went to Witley on November 26th, and on December 4th, while out with a walking party he left them, and was subsequently found at the railway hotel the worse for alcohol. He was brought back to London next day, but being a voluntary boarder was at liberty to leave. He left that day, and arrived at his destination without a ticket, having spent the money allowed him for his ticket on drink. We heard nothing more of him until December 12th, when we were informed that he was in Ipswich Gaol for having violently assaulted his wife with a candlestick, cutting her forehead.

Here was a person who was quite properly detained, both for his own chances of recovery, and for the protection of society. The law allows a lay person to step in, and upon his report the Commissioners in Lunacy have no option but to discharge the patient. There are those who would have the public believe that there are persons in asylums who are not insane, but the truth is that the tendency is rather towards discharging patients before they have become quite well. The sooner the law permits a person like this to be detained for some months in an institution, the better for themselves, their children, and society.

We will now return to *mania a potu*. If it is decided to treat the case at the patient's home, and it can often be done, especially if they are persons of private means, it is necessary that there

should be plenty of attendants. Choose a room on the ground floor, so that if the patient can go out there is not any struggling up and down staircases. Remove all unnecessary furniture from the room, and to prevent the windows being opened too far have them blocked, and the fire guarded. The patient's food must be minced fine, to obviate the need of a knife, and if the patient will not take food voluntarily, artificial feeding must be resorted to without delay. This, in private houses, is often a serious difficulty, especially where the patient is resistive, as the patient's friends object strongly to forcible feeding, thinking that the small quantity he may take is sufficient to maintain his strength. If that happens, and permission for his removal to an asylum will not be given, the only course for the medical man is to resign his connection with the case; if these people do not have food they go from bad to worse, and may develop acute delirious mania. In acute cases, Dr. Savage recommends 30 grains of trional, followed by an anæsthetic, the influence of which is maintained for twenty or thirty minutes. We have not yet given this treatment a trial at Bethlem, but Dr. Savage tells me that in his hands it has been attended with marked success, as the patient often sleeps for some hours, and wakes up quiet and composed. This method is certainly worth a trial, especially in private houses, because it is there that the difficulty is so great, on account of the lack of attendants.

I now come to *chronic* alcoholic insanity, which is commonly implied by the term alcoholic insanity. It is due to small quantities being taken often. Constant "nipping" is far more damaging to the brain and other organs of the body than bouts of heavy drinking. Derangements due to alcohol may produce a temporary disturbance of the intellect by means of its direct toxic influence on the brain; or, secondly, it may cause structural alterations of the brain, which are characterized by progressive weakening of the mental faculties, and finally, dementia. In chronic alcoholic insanity all the symptoms are less acute, and last longer than in acute alcoholism. Some persons drink for years without showing any signs of marked mental degeneration, though they may suffer severely from such disorders as cirrhosis of liver. Here, heredity often determines a person's fate.

It will be convenient to divide the symptoms

into sensory, motor, and intellectual. The derangements observed, whether motor or sensory, follow the law of dissolution of the nervous system, and proceed chronologically from the periphery to the centre, *i.e.*, from the least to the most organized.

Sensory disorders usually commence in the extremities of the limbs, and are often symmetrical. They may take the form of exaggeration, diminution, or perversion of general and special sensibility. Analgesia is common, and the patient may have a peculiar sensation about the skin, such as tingling or pricking, and these symptoms may cause delusions. The sensations are new to them, and, as in a normal subject, they want to know the cause; their seeking such cause with an already disordered mind brings them to a wrong conclusion. They misinterpret the sensations, and attribute them to mesmerism, hypnotism, electricity, and such like. Having once satisfied themselves of the cause, it is only a question of time before they weave ingenious tales as to who their persecutors are, why they do it, and how. Delusions are vague at first, and when patients can be got into an institution early, they can sometimes effectually be prevented from becoming organized and fixed. At first, patients have an idea that somebody is annoying them, but they are not able to say in what way; when they do arrive at the decision, and single out a person as the cause, it is then generally advisable to have the patient placed under proper supervision.

J. W. N., who is now in another asylum, presented a most interesting case. He first came here on August 29th, 1892, when he was 41 years of age, a commercial traveller, married, three children, the youngest of whom was *æt.* 5. This was the fourth attack, he having been in three other asylums, each time for a period of a month. The cause was alcohol, and there was also a history of heredity, his uncle and mother having been insane, and his youngest child was hydrocephalic; in addition, there was a family history of phthisis, and both father and mother were intemperate. His own history was that he had always been intemperate, and was in the employ of a large champagne firm. He had had a severe attack of influenza. It is interesting to note how often influenza or injury determines an attack of insanity in an alcoholic individual, or general paralysis in a person who has had syphilis.

In this case, the influenza occurred only a few months before the first attack of insanity. He was ill six weeks, delirious for three days, and had had two relapses. He had not been able to drink with the same impunity since. There was no history of syphilis, or of fits; he had had morning sickness, loss of appetite, and tremor. The previous attack had been of the same nature, and he was suspicious and violent. His present attack began three weeks ago; he became sleepless, depressed, suspicious, and very jealous of his wife. He thought his food was poisoned, and said he was afraid of doing violence to his wife and family. Hallucinations of all kinds were experienced. On admission his tongue was furred, and exhibited tremor, but his appetite was fair. Hallucinations of sight and hearing had disappeared, those of taste and smell remaining; he complained of a metallic taste, and smelt sulphur. Internal organs were healthy; pupils normal. He had a feeling of numbness in the tips of the fingers of both hands. Memory was fairly good, sleep fair, walk normal, and he looked healthy. There was a capillary dilatation on his cheeks.

He was discharged well on October 9th, 1892, three months after admission. He was re-admitted on November 9th the same year, and was said to have remained fairly well until October 20th, when he again became sleepless, and took to drink. He walked about the house at night fancying he heard noises, and gradually got worse. On admission he was incoherent, and wandered from subject to subject. He said he was a "switch," and "that every message on the telephone in the district went through him;" sometimes he would stop during a conversation, saying he could not answer, "as a message was going through;" and at other times would not answer questions, but would say, "we knew all about it." After leaving Bethlem in October he went to Margate, and when he returned home he told us that he "found his house had been rigged up with telephones, and could hear men making cat-calls through them at night." He would hum a tune, and say it "was caused by the telephone wires going through him." He fancied he saw the wires on his hands. At that time he smelt ammonia and chloride of sodium, as well as the fumes emitted by the batteries. Of course such hallucinations as these are traceable to education. The patient gradually improved, and was

discharged well on December 21st of that year. On December 30th, 1893, he left England to take a voyage to Australia for his health. I may say that, as a general rule, it is not good for these people to undertake a sea voyage, as, after they have been a few days on board, time hangs heavily on their hands, and they almost always commence drinking. This man booked to Melbourne, and had not taken drink on the voyage out; but, the summer being exceptionally hot, he took to intoxicants while there. On his journey home he had to be landed at Adelaide for a short time, owing to intense excitement, and on arrival in England he was brought to Bethlem. He had again conceived a dislike to his wife, and became suspicious about her; his hands and tongue were tremulous, he had hallucinations of all senses, and talked rapidly and in a wandering way, evading questions, but apparently remembered the details of his voyage. As time went on his delusions became more systematised; he said we had a system of "euphonic distribution" through the building, and that things were done to annoy him. He also said we had a system of sound carried by means of a needle apparatus, and he threatened to injure anyone whom he found carrying this about with him. He thought people used an unseen agency to produce visions of landscapes in his room at night. After that, things became more definite still; he said the attendants were the people who did it, and that they only did it because the medical staff ordered them to. Said they used "helioballs," "orophores," and "needle forms," and that he felt needles go into his head and then burst.

He remained here until June, 1894, when he was again discharged, but this time uncured, and a short time since he was still in an institution. So you see that after six attacks he finally develops organized hallucinations and delusions.

We will now deal with *motor* disorders. These often appear before the sensory. The finer adjustments become erratic, and the patient can no longer perform complicated movements. The tremor of an alcoholic person has the peculiarity that it decreases under the influence of alcoholic drink, and is most marked when the person is sober. The tremor is usually first noticed in the morning, and subsequently becomes manifest all day. The oscillations are regular and rapid, and

are exaggerated by voluntary movements. In consequence of tremor of facial muscles and the tongue, the speech is affected; there is no ataxy in the speech of alcoholics, such as in general paralytics, the speech being more blurred than hesitating. It is not uncommon to see paraplegia as a result of alcoholic poisoning, and the reflexes are also affected. Convulsions are due to organic disease, such as atheroma, softenings, hæmorrhages, though at times we have cases in which the fit closely resembles the epileptiform fit seen so commonly in general paralysis. These pass away rapidly, and the patient recovers, and leaves the hospital apparently well. The following case has an interesting bearing on this subject:—

H. H. A., æt. 32, a licensed victualler, was admitted into the hospital, February 13th, 1893, he had long been intemperate. The exciting cause here was an injury to the head; he had been in a railway accident in September, 1892, and his friends said that he had never been well since, nor had he been able to take the same amount of alcohol as he was accustomed to with impunity. There was no insane hereditary influence, but his father had been alcoholic. He had always been an excitable person, and on three occasions had had acute rheumatism. In September, 1892, just after the railway accident, he became irritable and uncertain, and the least amount of alcohol used to "fly to his head." On February 8th, 1893, he became intensely excited; he had then been drinking heavily for a fortnight. Family worries had troubled him, he had been sleeping badly, and refused solid food. He had attempted to cut his throat, and said he intended to commit suicide. He had hallucinations of sight, said people wanted to kill him, and that there were smells in his room. On admission, the left pupil was larger than the right, but both reacted normally, and the knee-jerks were exaggerated. Recent and remote memory were both bad; he was intensely excited, suicidal, and shouted to imaginary friends outside, who he said he could hear talking about him, and who would soon kill him. He asked for a razor so that he might cut his throat, and fancied he was at Kew. He had to be fed by means of the œsophageal tube.

On March 21st, he had an epileptiform fit, was deeply cyanosed, bit his tongue, and was stuporose for some hours. On the morning of March 22nd

he had another fit, was put on bromide of potassium, and in the evening was rather exalted. On April 6th, there had been no return of the convulsions, he was quieter, and took his food, and the hallucinations had disappeared. A week later he was again refusing food, and became more confused, and the week after that he had once more relapsed into a stuporous condition, and had to be fed by artificial means. On June 2nd, he had another general convulsion, falling suddenly, with complete loss of consciousness, and the stuporose state again followed. From that time he steadily improved, and within a month he went out on leave of absence. Six days later, however, he was readmitted on account of his excited state, but he rapidly improved, and left the institution well in August. He was again taken in on November 12th, 1894. He had remained well until eight days before admission, when he again became violent, heard voices, had hallucinations of sight, and tried to jump out of the window. He was brought in tied up, and would not answer questions; his tongue was furred, urine albuminous, pulse high tension, bowels constipated, and he refused food. He afterwards improved, and on January 9th was discharged recovered, and has remained well since. The great question about this patient was whether he was a general paralytic; but the length of time since the first attack, and the fact that he keeps well as long as he takes no spirits, probably indicate that the symptoms were all due to alcohol.

The *intellectual* disorders vary from mere irritability and restlessness to profound dementia and loss of memory. Loss of memory is one of the chief characteristics of chronic alcoholic insanity, and, as would be expected, the loss is greater for recent events than for remote ones; the patient can now no longer store fresh impressions. This is well shown by the following case:—

R. S. was admitted on December 2nd, 1895. His age is 62, and his occupation that of mantle-maker. He is married, and has ten children, the youngest of whom is 13, and the present attack is his first. He is said to have been ill a week, but it is probably a much longer period. He has been intemperate for several years. Fourteen years ago he was thrown out of a trap on to his head, and was rendered insensible for some hours. We cannot discover whether his drinking habits dated from that accident. Two months ago he

was connected with a law-suit, which he lost, and subsequently he procured a pistol and fired through the office-window in a random manner. Then it was noticed that his memory was rapidly failing—so much so that he did not know how to go about his own house, and failed to recognize his friends. He became extravagant, betted largely, and threatened his wife. Formerly he had excellent business connection all over London, but his failing memory appears to have deprived him of a large part of it. I will have him brought in, and will interrogate him in your presence. He is quiet and harmless, and, except for the fact that he cannot dress himself, and his dirty habits, we have no trouble with him. This is one of those cases which really might be treated at home if he had money, and could afford to have an attendant, or in the case of a pauper at the workhouse, and there are many such which needlessly fill up our county asylums. Many patients suffering from alcoholic insanity become suspicious, may misinterpret everything, and they often do mistake identity, speaking to a stranger as though he had been on familiar terms with them. Hallucinations of hearing, and, indeed, of all the special senses, are common. Sooner or later, delusions of persecution appear, and, as a result, acts of violence may be committed. Whether a person becomes homicidal or suicidal varies with the individual. One patient will retaliate upon those whom he deems his persecutors, while one who is more peaceably inclined will prefer suicide. Another interesting class of persons are those who believe they are constantly being watched and being talked about in the streets; these may develop ideas of exaltation, their argument being that they must be important personages to arrest so much attention. Women constitute the majority of these latter cases, some of them being governesses who have to work to maintain themselves.

Some cases of alcoholic insanity are of melancholic type; they refuse food, and are commonly suicidal; other cases, again, may be either maniacal or stuporous. Some from the first are apparently weak-minded; such are dirty in their habits, untidy, and, the most marked symptom of all, their loss of memory is great.

I will now show you a patient, H. G., æt. 60, who considers he has been persecuted; his occupation is that of proof-reader. He alleges that

people in the institution have stolen several of his watches, as well as a coat. He also has stated that the Queen has been staying at his house. His memory is very defective, he cannot remember names of people about the hospital, nor dates: like the last case we saw, is quiet and apathetic.

Another patient whom I will show you is 56 years of age, married; one child, æt. 22. He is a wine merchant. This is his second attack, and he has been here two months. Alcohol is the cause. His father was in Bethlem Hospital; elder sister also insane. (This patient was interrogated at length, and his replies showed him to be unaware of many events which happened prior to admission. Questioned as to his drinking habits, he acknowledged that he had been addicted to intemperance.) Before admission he thought that his food was poisoned. His tremor is very much less than when he came in, and his tongue, which on admission was red and raw, is now fairly healthy. He is still quarrelsome with other patients.

As to diagnosis, chronic alcoholic insanity has to be diagnosed from such disorders as general paralysis, lead paralysis, acute mania, morphia and cocaine palsy. It is often most difficult, and sometimes impossible to say whether a person is merely suffering from alcoholic insanity, or whether it is a case of general paralysis. In both there is loss of expression, tremor, and there may be inequality of pupils, and convulsions. In coming to a decision on a case, the past history may assist you; also the character of the speech. Again, an alcoholic has anæsthesia more marked in the lower limbs than a general paralytic. The presence of hallucinations has also a certain value, for, though not uncommon in general paralytics, they are much more frequently met with in alcoholics.

Lead palsy, like that due to alcohol, follows the ordinary laws of dissolution of the nervous system. The tremor is usually slow, slow ideation, dread, hallucinations of sight and hearing, often furious and maniacal delirium. They have rapid remissions and recurrences of the delirium. Then there are the somatic symptoms—colic, the blue line on the gums, paralysis of extensor muscles. Assistance is afforded in diagnosis by the occupation of the individual.

With regard to morphia and cocaine, they produce intense hallucinatory delirium. Morphia

alone seldom produces hallucinations, but cocaine brings on vivid ocular hallucinations, but these do not cause alarm as in delirium tremens, there is also marked disturbance of general sensibility. If there is difficulty in diagnosis, temporary use of the drug usually re-establishes the mental equilibrium. A large percentage of morphia cases come into asylums, and they are only too commonly medical men; of the last four admitted, two were medical men, and one a medical student. One such gentleman in at present is very interesting; he believes himself to be the subject of hypnosis.

As regards chloral, the same class of symptoms are not met with as in alcohol. I may just remark that patients often come to medical men saying that one glass of alcohol produces intense flushing of the face. This symptom is not of much importance in women, but it may be in men. Where you find this, it is always well to ask if the person is sleeping well. Frequently, you will learn that they are not, and you can then ask what they are taking to induce sleep. Chloral is often at the bottom of the complaint. The drug also produces depression, and gastric disturbance.

The next patient I will show you is, I fear, a general paralytic. G. H. B. was admitted on February 8th this year; æt. 34, occupation, inland revenue officer. This is his first attack, and he is said to have been ill a week before admission. He is neither suicidal nor dangerous, but has been intemperate, and there is also a history of syphilis. The family history is said to be good. Two years ago he was struck on the head by a cricket ball. In December, 1894, he was noticed to be duller than usual, and this was more marked in July last. Therefore, you see that instead of being a week ill, as the date given by the friends, we have a history extending over a year. He complained of nothing particular, but was extravagant and boastful, and later on he developed hallucinations and insomnia, and asked his brother to buy him prussic acid. He also threatened to shoot persons. On admission, he had tremor of tongue. He has convergent strabismus and myopia. The right pupil is larger than the left, they both react to light, and he can squeeze 85½ lbs. His gait is unsteady, and his speech slow and hesitating. There is marked loss of expression. He has heard singing, and, later on, voices, but they do not persist now so much as to disturb him; they were always on

the left side of the head. He said he used to hear John the Baptist telling him he was a fool, and could see things in his room, but they did not appear to be panoramic. The knee-jerks are much exaggerated; there is distinct tremor of the face, the patient attributing that to the oxygen which he tastes and smells.

I now wish to allude briefly to dipsomania. We have seen that some persons drink because their moral sense is defective; either because it has been feebly developed, or because it has become obliterated. Others, again, indulge through weakness of will. So-called dipsomaniacs are not common drunkards, and must not be so regarded. They are persons who suffer from a periodic impulsive form of insanity, which manifests itself in an imperious craving for alcohol or some other drug. That dipsomania is closely allied to other forms of periodic insanity is shown by their inheritance, and by the fact that often members of the same family have attacks of mania, melancholia, etc. These individuals are ashamed of their degraded habits, and are perpetually striving against them. The impulse to drink comes on in attacks, and between the attacks no alcohol may be imbibed, the persons becoming, during this period, useful members of society. The initial stage is usually short, and may vary from a few hours to a few days; during this period their mental anguish is shown by depression, restlessness, anorexia, loss of attention, and sleeplessness. When the desire for drink arises he may confide in a friend and ask his help, or may lock himself up in a room. When once he has fallen, he obtains alcohol by all possible means, and will even steal it. He may drink for some days, until he exhausts himself or develops delirium tremens. On recovery, he may remain well for a long period, and, if looked after, may never have a relapse. Unfortunately, however, it is more common for the attacks to be frequent. Some become so ashamed of themselves after an attack that they commit suicide. In dipsomania, alcohol is a complication, not a cause, and alcohol may be replaced by some other drug, such as morphia or cocaine. An ordinary inebriate differs from a dipsomaniac in several respects; the former drinks from vice or defective moral sense, and is indifferent about it. Moreover, he does not strive against the habit, while a dipsomaniac has to gratify an imperious, impulsive desire. He hates

alcohol, and strives against it, and he is ashamed of his condition.

The treatment of dipsomaniacs is to isolate them and remove all alcoholic beverages. Bitter drinks should be given, and care must be exercised in giving drugs for insomnia, as, in overcoming one habit, another may be formed. Never give morphia or cocaine, and, whatever drug be used, be careful to change it from time to time, so as to avoid the formation of a habit. When the attack is over, remember that prophylaxis is better than cure, so that care must be taken to prevent further attacks.

Regarding prognosis: in giving a forecast of a case of alcoholic insanity, it is desirable to divide it into (a) immediate and (b) ultimate. A certain eminent physician, when discussing the immediate chances of recovery of inebriates, often says, "To the alcoholic all things are possible," meaning thereby that however grave a case may appear to be, it is never safe to give a bad prognosis. Of course this refers chiefly to the acuter forms. We have had cases here in almost the last stages of the disease, which have got entirely well, so that the axiom I have just referred to is a very useful one. A patient may get well five or six times, but after many relapses the memory goes, or systematized delusions may develop. Therefore the immediate prognosis of an acute case, though frequently favourable, should be guarded, because each attack leaves the patient mentally weaker.

The treatment of alcoholic insanity I will divide into (1) prophylactic, (2) curative. In the first place, prophylactic treatment, in the present state of the law, is most difficult. The temperance societies have done much good, but they only assist those who are willing to be helped. True, the Inebriate Act allows persons so afflicted to be detained, but only after they have themselves signed a document, as required by law, and in which the period of detention is specified. Naturally, it is no easy matter to persuade a person who is already weak and vacillating through drink to sign such a document. No really efficient service will be done until the law permits the enforced detention of drunkards and other alcoholics in institutions for a considerable period. These institutions ought not to be furnished with the comforts of modern asylums, but should be more of a prison type. I think such will come about sooner or later. The

evils wrought by these individuals are not only the ruining of their own future and spoiling the happiness of those about them, but they beget degenerates, and it is from alcoholic stock that our asylums are largely filled.

As regards curative treatment, you have first to settle whether a particular case shall be treated at home or in an institution. If the patient have means and exhibit merely a loss of memory and untidy habits, there is no need to send him to an institution. If, on the other hand, hallucinations be present, especially if they be organized and there be suspicion of certain persons, the patient should be placed under proper control. When they come into institutions, of course, the first thing to look to is their food. Because they have chronic gastritis and irritable mucous membranes, is no reason why you should not feed them. The man, æt. 56, whom I showed to you, had, on admission, an intensely red tongue, and had had constant sickness. We made him take daily about six eggs, three pints of milk, two pints of beef tea, etc., and vomiting did not occur afterwards. Alcohol must be entirely removed from the dietary; we act similarly with morphia habit. An uncomfortable day or two will be experienced, but when once the habit has been broken that trouble is over. There are drugs which can be used for sleeplessness, but they are of very little use in these alcoholic individuals. Lastly, they should be detained as long as possible, so as to ensure convalescence; otherwise a relapse is to be feared.

A POST-GRADUATE LECTURE

ON

AURAL POLYPI.

Their Symptoms, Diagnosis and Treatment.

By GRIFFITH CHARLES WILKIN, M.R.C.S.

CLINICALLY these growths may be described under three heads:—

- (a) The flesh-like aural polypi, which may be either soft or firm.
- (b) The gelatinous aural polypi, sometimes spoken of as myxomatous.
- (c) The malignant aural polypi.

Microscopically the first division consists of fibrocellular tissue, plus a few blood vessels, etc., and is covered as a rule with layers of stratified squamous epithelium.

The second division, consisting of polypi, which cannot, by the naked eye, be told from true myxomatous growths, is found microscopically to consist of a somewhat similar ground substance to the above, studded with large lymph spaces, and sometimes these tumours are said to have a true adenomatous character. The third division, the malignant aural polypi, are fortunately very rare. They may be either sarcomata or carcinomata. The microscopy does not differ from that of growths of the same nature found in other parts. Papillomata rarely occur in the middle ear, but are occasionally met with in the external ear. True granulation tissue frequently occurs.

Etiology. In almost all cases of aural polypi, one finds a history of prolonged discharge, and generally of hæmorrhage. There are, however, authentic cases where the polyp was apparently the primary disease. Grüber mentions the fact that Moos removed a polyp growing from the posterior part of the tympanic cavity, and bulging the membrana tympani outwards. To remove this, the membrane had to be cut. Other cases are also on record.

Symptoms. Subjective. If the polyp is small, the subjective symptoms, supposing the growth to be benign, are very similar to those of chronic suppurative disease of the middle ear, plus occasional slight attacks of bleeding. When obstruction to the exit of discharge occurs, great pain will be set up in the ear and head, as well as severe tinnitus and giddiness.

Objective. If large, the growth may be seen protruding out of the external meatus. I have seen one that filled the whole of the concha. When it is small the secretion requires to be removed, and then, by aid of a speculum, the tumour becomes visible as a red, or possibly, gelatinous mass in the canal. With a probe it may easily be recognized from an exostosis, with which it otherwise might be confounded, by its free movement and soft consistency. At times the seat of origin can be ascertained by moving the probe freely round the polyp. In some cases the use of the probe may cause smart hæmorrhage.

Prognosis. Benign polypi, when removed, and

their bases thoroughly treated afterwards, are fairly amenable. Though, when a large area of bare bone is found, the after-treatment is much more prolonged, and recovery far more uncertain. A guarded prognosis is always advisable until the tumour has been examined carefully by the aid of the microscope.

Should there be any question about the advisability of removal when an absolute cure cannot be promised beforehand, the answer is, the removal is absolutely necessary, because, as the polyp increases in size it closes the meatus more and more, thus preventing a free outward escape for the discharge, and so being a possible cause of mastoiditis and its consequences. As a rule polypi depend for their origin on a condition of prolonged suppuration, and when once formed the presence of the polyp increases the discharge. All cases, then, of suppurative otitis must be looked on as a distinct danger to the life of a patient, and should be arrested as quickly as possible. The old idea that a discharge from the ear must be left alone from fear of something worse ensuing on its arrest, is, I hope, for the sake of the public, absolutely dead. In all cases of aural polypi the nose and post-nasal space should be carefully examined. In children and young adults post-nasal growths are by no means infrequently found associated with them, and nasal trouble is a frequent concomitant.

Treatment. Having an aural polyp to deal with, the only good treatment is removal. This may be done either by ring-knives, forceps or snare. For the sort of instrument to be used the operator must be guided by three things: (1) the size of the growth to be removed, (2) its position, (3) by his own custom. In very large, as in very small polypi, the forceps is, in my opinion, the best instrument; for medium sized growths some form of snare may be used. For mere granulation tissue, which resists medical treatment, a ring-knife may be used, especially when the tissue is on the lateral wall of the meatus.

Supposing a snare is chosen, the next point is to decide on the nature of the wire. Piano wire, as used with the nasal snare, is, of course, on account of its spring undesirable. Silver or iron wires may however be used, and meet all the requirements, viz., both are sufficiently strong to

detach the polyp from its point of origin, and are flexible without spring. The strength required is not very great, so that the finest wire may safely be used in iron, but though as a rule, I should give the preference to silver when a very fine thread is required, it is not reliable. It may be thought that with cocaine the spring of piano wire would not matter, but unfortunately, when a polyp is present cocaine cannot satisfactorily be applied to the external auditory canal. If you can make out the attachment of the polyp with a probe, do so, but in many cases this is almost impossible. In such cases, the best way to use the snare is to pass the loop over and round, and the barrel under the growth, carrying the point as near to the base as possible. Then tighten the loop firmly and steadily until the polyp is completely cut through. As a rule, the growth comes from the meatus with the snare when withdrawn, but at times, especially with small polypi, it remains in the meatus. It can, however, be easily removed with the syringe.

For arresting hæmorrhage, which is generally very free, gentle syringing with hot water usually succeeds, or the insufflation of finely powdered alum, which has the double effect of arresting the bleeding and shrivelling up the base, may be tried. Hazeline is a useful styptic to have by one for these cases. After the ear has been carefully syringed out, and the bleeding stopped, the operator should always examine again, and, if need be, a second operation may be done at once. For after-treatment, a solution of boracic acid in rectified spirit and water may be used, beginning with half and half, and gradually increasing the proportion of the spirit. Alum may also be used, and is said to be very good. The ear must be syringed out at least once a day with some antiseptic lotion.

For local application it will be generally found necessary to use either chromic acid, solid nitrate of silver, or the fine cautery point. Should the pain be very great after the operation, douching with hot water, or leeching in front of the tragus gives relief. Should removal be objected to, caustics or astringents have been recommended in one form or another—nitrate of silver, solid or in strong solution, sulphate or acetate of zinc, perchloride of iron, acetate of lead, creasote, nitric acid, sulphuric acid, lime water, and sulphate of copper. Toynbee recommends a mixture of quicklime and caustic potash, to which a little sulphate

of iron is added, to prevent rapid deliquescence by which the healthy parts would also be attacked. Chromic acid, recommended by Seely in 1872, is also one of the most effective caustics. A crystal is to be taken up on the moistened end of a probe and applied to the growth, the process being repeated after the eschar formed has separated. Its action is more energetic than that of nitrate of silver, but sometimes causes inflammation of the canal, even when applied most carefully. Politzer recommends perchloride of iron, in crystal or solution. A piece of cotton wool is to be dipped into the solution and applied to the growth, or a crystal carried to it by means of a probe. Grüber does not speak favourably of this, or the alcohol treatment, praised also by Politzer. The translators of Grüber's work into English, however, have found this latter treatment useful, when operative treatment is impossible. At the same time, the following significant remark was made: "The method is naturally only applicable in the case of such flabby polypi (granulations) not associated with bone disease." Lactic acid (15-20 per cent. solution) has been recommended by Victor Lange in cases of polypoid proliferation. I have given you the opinion of others on the treatment of aural polypi without removal, because there may arise circumstances which absolutely preclude operative interference, but as a rule to allow an aural polyp to remain in the ear is, for the reasons I have before given, decidedly bad surgery.

The remaining cases of aural polypi may be generally referred to two sorts of malignant growths, viz., the sarcomata and epitheliomata.

Sarcomata are said to be primarily developed in the auditory canal, and rapidly extend to the neighbouring structures. Of course they may equally readily involve the ear from the neighbouring parts, but in such cases they would hardly come under the heading of aural polypi, nor would they probably come under the notice of the aural surgeon. The fibro-sarcomata are said to remain for years without causing any particular symptoms. On the other hand, the small round-celled sarcomata, growing very rapidly, set up a great amount of pain, are generally, if not always, associated with a foul-smelling discharge, more or less sanguineous. The surrounding parts are rapidly implicated. If seen and recognized early enough, it might be possible to remove the growth with the

knife. The lack of recognition appears to me due to the fact that growths removed from the ear were, at least formerly, not thought worthy of microscopical examination; and, secondly, people suffering from aural disease frequently seem to think that nothing can be done for it, and leave the trouble to treat itself until some more offensive symptom than usual drives them to the medical man for relief. Epitheliomata are said to occur after prolonged suppurative otitis. The commencement differs in no way here from that in other parts. Pain is not such a constant symptom as in the rapidly-growing forms of sarcomata.

A case of malignant growth of the left ear, which came under my care some time ago, I will briefly relate to you, because it illustrates strikingly the necessity for microscopical examination in all cases of growths removed from the ear, and because the treatment which I adopted is comparatively new, in this country at least.

When I first saw the patient, a woman, she was suffering from great pain in the left ear, which was said to have come on after the removal of a growth a few weeks previously at another hospital. The auricle was standing out from the head, and there was general tenderness round it, a soft brawny swelling in front, and a greatly enlarged mastoid process behind, over which the skin was adherent and discoloured. The external meatus was almost completely closed by an œdematous-looking swelling of the walls of the canal. Some of this tissue was removed and examined microscopically. The result pronounced the growth a squamous-celled epithelioma. Clinically the growth had appeared much more like a sarcoma. The woman said that about a year previously she had been knocked down by a cab when crossing the road. The whole of her left side had been severely bruised, and two of her ribs broken. The left ear and side of her head had been bruised and cut. Some three months after this she was compelled to go to a hospital for treatment, on account of discharge from the left ear, with deafness and tinnitus. For some months she remained an out-patient of the aural department, after which, not having received much benefit, as she thought, for a time she appears to have given up treatment. Then she went to another hospital, where a growth was removed from the ear. This gave her so much pain that she would not return,

and a few weeks later she was sent to me. In my opinion the growth should have been microscopied at once, and had this been done the patient would not have been sent away. It is quite possible to get a fair idea of the specimen structurally from a fresh section made at the time of removal, and in the case of out-patients, I think that every large hospital should do this. When I received the microscopical report, and thus learnt the nature of the growth I had to deal with, I thought of Von Mosetig's treatment of malignant growths, and seeing the evident signs of mastoid involvement, together with the anterior growth, I determined to try it, with the consent of the patient. Possibly, you may remember Professor Von Mosetig, of Vienna, bringing before the Vienna physicians several cases of malignant and benign growths which he had treated by parenchymatous injections of methyl violet or, as it is now called, pyoktanin. His report on this was so favourable, that, seeing how hopeless this case was for surgical treatment, I determined to give it a trial.

In this case I commenced with a solution of 1-500, injecting in different parts of the growth from 10-20 in all each day for ten days, then I raised the strength to 1-300, and then to 1-100. The action of the drug was quickly visible; the growth contracted and became very hard, after about a fortnight, a soft apparently fluctuating mass gradually replaced the harder growth. There was no rise in temperature. On opening the soft mass, I found a gelatinous violet substance round the hardened growth. This process of contraction went on whenever the pyoktanin was used, and although the growth had advanced too deeply for me to hope to save the patient, I was quite satisfied with the action of the drug. It acted remarkably in relieving the pain, which alone would make it a drug worth trying. I constantly examined the urine to see whether it was affecting the system generally, and especially the kidneys. I never found any albumen or sugar, the quantity of urea passed remained about the same. The urine from being very foul smelling when the patient was first admitted into the hospital, became quite sweet while the injections were used.

After death, which took place sixty-two days after the commencement of the treatment, the growth in front of the ear was examined micro-

scopically, the report said that no epithelial cells were present, and that the thickening was due to fibrous tissue. As this was the only part that could be properly attacked, the result was extremely good. The external meatus and the mastoid process formed one large cavity, caused by the action of the drug. A very thin plate of bone existed between the dura mater and the upper part of the cavity.

A second case has since come under my treatment. The growth differed greatly from that in the above, being very much harder. It appeared first as an aural polyp, red and rough-looking. At the base of this was a considerable area of bare bone. In front of the ear was a mass of considerable size. The mastoid region was not apparently involved. The case was handed over to me by my colleague, Dr. Stoker, who, after removing the growth in the ordinary way found it recur. The great density of the growth prevented much fluid being used at a time, until it gradually became softer. Without any direct treatment to the aural polyp, it gradually shrank up as the injections were carried on. The patient has passed out of my hands for a time, but is, I believe, still alive and well.

PENTAL.

By DUDLEY BUXTON, M.D., B.S., M.R.C.P.,

Anæsthetist in University College Hospital, etc.

ALL new anæsthetics should obey two canons; (1) they should be comparatively free from danger to life, and (2) they should possess special advantages over similar bodies. Pental, as it is used at present, is associated with a certain amount of danger. We are, however, quite prepared to alter this judgment, and to learn that when the drug is further used—perhaps somewhat differently—it may meet with more success than up to the present has been realised.

It may be useful to consider the mortality under pental. An English observer reports one fatal case out of 149 administrations (in this series of

cases, however, it seems possible that this death was not solely due to pental); according to statistics, Gurlt gives from German sources three deaths in 600 administrations; Snow gives two fatal cases in the course of 238 administrations; Wagner one in 199 cases; while there may be other cases which have not been recorded. It must further be borne in mind that in many cases untoward symptoms have occurred. Thus we get a mortality of 1 in 190. On the other hand, Rûth records 2,131 cases, and Phillip 1,000, without a death; while Rûth, who appears to have had a pretty wide experience of the drug, speaks highly in its favour.

In dealing with statistics, it is necessary to be sure of our ground, and to closely examine these reported deaths from pental. In the case of Snow's deaths, it was stated by Thudichum that the preparation he employed was impure, containing intermediate hydrocarbons. One of the many difficulties in working with pental is the liability to this risk of impurities. Prof. Dastre in his book speaks of amylene, or as we should call it, pental, as having "fallen completely into desuetude owing to its disagreeable odour." It is clear, therefore, that he speaks of a substance which is an impure drug, a mixture of various hydrocarbons with which we are now quite familiar, having apparently the same formula and the same constituent molecules. Before pental can be justly accused of causing death, it must first be clearly ascertained whether the substance employed was pure pental, and not some mixture simulating it. How, then, is it possible to be certain of the material used? It may be said that there is safety in going to reliable firms of manufacturers, but that is not so; it is known, as a matter of fact, that impure samples of drugs may be turned out from the manufactories. For instance, leading manufacturers of chloroform will sometimes supply more than one quality, so it is clear that there is no safeguard by that means against impurity; and possibly much of the pental used up to now, and as at present supplied, is not absolutely pure. There is no ready test that can be applied to it; the test of smell is perhaps the most reliable, but to really arrive at a just appreciation of the quality of the drug, it would be necessary to go through an elaborate chemical analysis, perhaps taking a great deal of time, and requiring an intimate knowledge

of technical chemistry, which in many cases might be wanting. Pental is alleged to have certain disadvantages. For example, Kleindienst has found albuminuria occurring in many, and hæmaturia or hæmoglobinuria in several cases within three or four days after the administration of pental. These, of course, are very troublesome symptoms, and in many cases would be very untoward and difficult to satisfactorily explain to the patient.

Records must vary according to the different opportunities possessed by the observers. Thus, one series of observations deal only with the very brief anæsthesia required for the extraction of teeth, others record longer periods of anæsthesia under pental, noticing, in consequence, more after-effects. One observer, who has used pental largely for children in a Viennese hospital, with a record covering over a large number of cases, speaks of it in glowing terms as being the anæsthetic *par excellence* for children, but states that he finds cyanosis not uncommon, and clonic spasms of frequent occurrence. He records only one instance of albuminuria in 1,000 cases of the use of pental. In the opinion of this observer cessation of respiration and cyanosis are due, not to the inhalation of pental, but to the results of the clonic spasms of the muscles of respiration, arising from the action of the anæsthetic on the nervous centres. It is most desirable that, in all cases of death under the administration of pental, it should be clearly stated whether anything in the nature of a spasm occurs. In one case there has been noted spasm of the glottis and diaphragm. In the very careful work of Dr. David Cerna he showed that in lower animals you get a very marked fall in arterial blood pressure, but whether that fall in blood pressure is important, time alone will show for certain. If it be associated with weakening of the heart muscle and want of tone of the arteries, then pental is an anæsthetic similar to chloroform, possessing its dangers and being, perhaps, less manageable. Snow held the view that amylene acted upon the heart and killed through failure of the circulation. In a paper written by Dr. Holländer, that observer speaks of a condition of what one might call somnambulism, occurring with the administration of pental, the patients becoming analgesic and, on being told to open their mouths, doing so and making no resistance during the extraction of teeth, yet re-

maining all the time unconscious of pain. Snow refers to a similar state, and used it in obstetric practice. All observers do not mention that their patients are equally complaisant, but it really would be a most desirable thing to possess an anæsthetic rendering patients so obedient, at the same time causing them to be unconscious of pain. Dr. Holländer also speaks of the frequent occurrence of clonic spasms and of rigidity under pental. The muscles of the jaw seem to be particularly affected, so much so that in some cases it is not possible to get the patient's mouth open to perform any necessary operation. Taking such records as there are of what may be called the induction period, the variation of its duration is very noticeable. Of this there are two possible explanations: one is that the patient's individual peculiarities may account for this wide range of difference and the other is that the drug, as now supplied, is not only marred by impurity, but that the degree of impurity is variable. Another important point in connection with the administration of pental is the question of its liability to rapid decomposition, and until this question is definitely settled caution is advisable in the choice of this anæsthetic.

To sum up, then, pental, as far as is known, is a most useful drug, but it possibly and probably possesses grave disadvantages from its liability to affect the respiration, doing so possibly through spasm of the glottis and diaphragm. In the cases of death it is to be noted that they have arisen from failure of respiration, while the heart has continued beating for some minutes afterwards. It is very easy to do a marked injustice to any new anæsthetic if, through cases of death occurring in the early days of its administration, its use is, therefore, discarded. It is possible, perhaps, that that death may be one in a million, though, on the other hand, it may be one in a hundred. Provided untoward symptoms jeopardising the patient's life do not present themselves frequently, it seems justifiable to continue the use of a drug, even if in the early days of its administration a few accidents have occurred, provided it can be reasonably demonstrated that such fatalities are not a necessary or a frequent result of its use.

THERAPEUTICAL NOTES.

Chloroform or Æther.—Prof. Mikulicz (Breslau) has returned to the practice of administering chloroform after giving an extended trial to ether, and gives the following (summarised) reason for his preference for chloroform. He states that, although he had no actual death while the subject was under ether, he experienced during the anæsthesia, but more especially after the return of consciousness, accidents of several kinds by no means harmless. The selection of cases was carried out most strictly; all patients with pulmonary disease, very young children, old weakly persons, patients with heart disease or severe anæmia were excluded. Notwithstanding this, out of eighty examples of ether anæsthesia, he met with three cases of threatening asphyxia during the administration, two of collapse after recovery, four of acute bronchitis, and two of ether-pneumonia, with œdema of the lung. From these facts he draws the two conclusions—(1) that ether cannot be looked upon as safer than chloroform; (2) that every administration of an anæsthetic must be looked upon as having a risk of its own, and, consequently, grave consideration must be given to the question, Is general anæsthesia necessary?
(*Berl. Klin. Wochens.*)

Lysidin for Gout.—Staff-Surgeon E. Grawitz has lately tried lysidin, a new remedy for gout, which is said to have five times the strength of piperazin as a solvent for uric acid. He used it in increasing doses, beginning with 1 and going up to 5 grams as the daily quantum, dissolved in soda water. The results he obtained were most satisfactory, several tophi disappearing under its use.
(*Deutsche Med. Woch.*)

Diabetes Mellitus.—The thirst is said to be greatly relieved by Pilocarpin, given as follows:—

R. Pilocarpin nitrate	...	05 gram.
Spirit. vini gall.	...	3'0 "
Aq. dest.	...	8'0 "

Sig.: The tongue to be moistened four or five times a day with a few drops of the solution.

(*Nouv. Remed.*)

Antigalactagogue.—Painting the breast with a 5 per cent. solution of cocaine proved very useful in checking the milk secretion in a woman whose child had died. No unpleasant effects resulted from the application.

(*Wiener Medic. Woch.*)

Prurigo.—Simple coal tar without modification is reported to be particularly useful—much more so than its artificially separated components—in prurigo, dry eczema, psoriasis, etc. The following is the prescription:—

Tar simplex	...	3 parts
Spirit (95 %)	...	1 part
Sulphuric ether	...	2 parts

The application can be easily removed by olive oil.
(*Monatsch. für Dermatol.*)

REVIEW.

Diseases of Children. By ASHBY & WRIGHT.
Third Edition. (Longmans & Co.).

Price 25s.

This book has been steadily forging its way to the front as the book on children's diseases, and the present edition will help it still farther. The work has been very carefully revised, and brought up to date by numerous additions; the bulk of the book still increases, notwithstanding the large amount of smaller type used. We have tested the information given in several cases, and have always found it practical and to the point. The summary of the principal errors, for instance, which may be made in a diagnosis of tubercular meningitis; a short article on ascites; one on enlarged spleen—these are all excellent. The appendix, containing articles on milk, and other dietary for ailing childhood on general therapeutical principles, poultices, baths, purgatives, etc., strikes us as one of the best specimens of its class we have ever read. Of the formulæ at the end of the book we can speak most highly, from personal experience of many of them. We have little doubt that the book will continue to have a large and increasing sale.

THE CLINICAL JOURNAL.

WEDNESDAY, APRIL 1, 1896.

POST-GRADUATE LECTURE

ON SOME POINTS IN THE

DIAGNOSIS AND MANAGEMENT OF HIP-JOINT DISEASE.

Delivered at the Central London Sick Asylum, March 12th,
1896.

By RICHARD BARWELL, F.R.C.S.

IN an infirmary such as this we rarely meet with cases of recent and rare disease, such as we find in our hospitals or like institutions, where frequently the only difficulty as to the subject of a clinical lecture is the choice among many interesting forms of disease. Nevertheless we meet here a number of cases, not at all uncommon, but rather of daily occurrence; and I am not sure but that such cases may form, for the family practitioner, a more useful subject of discourse than diseases which the medical man may perhaps see once or twice in a lifetime, or, even more commonly, may never see at all. Now among these very usual cases Dr. Hopkins has been good enough to show me three children with hip disease—a malady, which has been written about and talked about so much, that I may possibly be unable to say anything on the subject that is absolutely new, although I believe it likely that I shall be able to refer to certain points in the diagnosis and treatment, which, though I and others have insisted on them, still remain in a certain degree unrecognized. I hope to be able to interest you on these points.

Hip disease, although it may occasionally begin later in life, is essentially a disease of childhood, even of infancy—that is, it commences at a period when the patient can give very little information about its troubles; and yet it is important to recognize the malady in its very earliest stage. The mother or nurse says the child cries when being washed on her lap, especially if she move its legs; it, if old enough, calls out, “Oh my

knee!” and grasps the limb just above that joint. It may be, too, that the child, if of an age to walk, limps or drags a limb, and it very soon gets tired, and cries to be taken home. This is often the only information procurable.

But these are subjective, we want for a clear diagnosis some appearances that we can perceive and verify for ourselves. Too much stress must not be laid upon the systematic division, into first, second and third stages. I have seen many cases in which lengthening or shortening have been absent for weeks after commencement of disease—neither is knee-pain, nor indeed pain at any definite spot invariably present. The old plan of seizing the child’s thigh, and jerking the bone upward into the acetabulum is most barbarous, may be very injurious, and has besides this disadvantage, it not only hurts but also frightens the child. Now, if a child come to regard the doctor as an object of suspicion and fear, as a bringer of pain and terror, that child will only scream and sob at his approach. He can never elicit any information from gesture, posture, or by word of mouth. If on the other hand he be gentle in manipulation and in manner, he will in a little while be able to manage the most recalcitrant.

The first symptom is that which in my work on *Diseases of the Joints*, I have called fixity or immobility of the thigh; it precedes, certainly in very young children, even a limp, nevertheless, as you will more generally be called to children who are old enough to walk, and whose parents are disquieted by the lameness, I should first like to describe the characteristic limp of hip disease. You will probably sit down, let the child, quite stripped, stand near, and with his back to you, tell him to walk straight ahead—remember that as I told you the thigh is fixed, and the hip-joint is tender. Thus, the child never advances the right (the affected) limb in front, or, but very little in front of the other, also, he lets his weight fall for as short a time as possible on the diseased joint. You will then perceive an abnormal motion of the buttock and pelvis on the diseased side, in order to let the limb come forward, and you will both

see and hear an altered rhythm of the step—they fall in what a musician would call *dotted notes*—there is one long accentuated step, and one short, slightly marked one alternately. Also, when the right foot bears the body-weight, the trunk sways over to that side. (NOTE.—To save words, the *right* is taken as the diseased hip.)

This first examination causes you to suspect, but only to suspect, not to ascertain hip mischief. To do that quite early in the case you must verify “fixity of the thigh” in the following manner: Place the child on its back, with the limbs straight down; quite early in the disease it can lie thus, with the loins straight and in contact with the couch. Tell it to lift up one leg (I am assuming you do not as yet know which is the diseased side), and watch the position and action of the pelvis; if that bone remain still while the leg and foot are raised to about the perpendicular, direct the child to put that limb down again on the couch, and to raise the other. If there be any hip-mischief at all we may be quite sure that the child’s first choice of limb has fallen on the sound one; watch, therefore, the second one with the more care; you will see that in raising it he does not really flex the hip-joint at all, but moves the whole flank and pelvis; you will notice that the crest of the ilium, the tuber ischii, etc., follow the movement or rather the apparent movement of the thigh; neither will he lift that limb to anything like the angle of the other. After this take the thigh in your hand and very gently, as I show you on this patient, flex, ad- and abduct it; you perceive that every movement is so followed by the pelvis that the hip-joint is, in reality, still. This is what I have termed “fixity of the thigh;” it is brought about by contraction and rigidity of, among others, the capsular muscles. This rigidity is partly reflex, partly emotional, therefore it is entirely annulled, in all the earlier phases, by an anæsthetic, and has nothing whatever to do with ankylosis. What are the resultant evils of that muscular contraction I will tell you presently.

Having now ascertained the presence of hip disease, you in watching and treating the case will, if it still progress, observe changes in the condition of limb which go by the name respectively of lengthening and shortening—the former usually, though not always, precedes the latter. These differences in the length of limb are apparent only,

and depend upon the fact that the above-described fixity of thigh occurs first in the position of abduction, and then of adduction. I cannot say why the reflected nerve-irritation should at one period be thrown on the gluteal, and at another on the obturator nerve. I will, however, show you how abduction produces what is called lengthening, adduction shortening.

Here is a pelvis with the femora; I abduct the right one. But a child will neither stand nor lie with

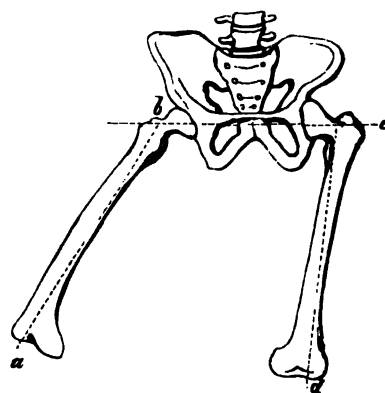


Fig. 1.

one thigh thus projected to the side; he will make the thighs lie parallel. To attain this he must droop that side of the pelvis, the transverse axis becomes oblique—in other words, the acetabulum of the

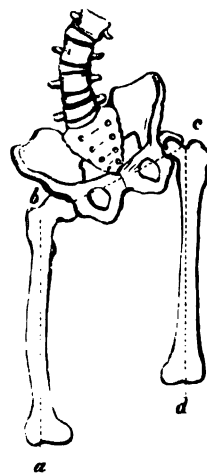


Fig. 2.

abducted side lies below the level of the other—therefore, the limb *seems* lengthened—it is, however, only apparent lengthening. Take the contrary side of this question, viz., the result of adduction.

I place the left femur in that position. The child, however, will not lie with its legs perpetually crossed, but will get the thighs parallel by raising the diseased side of the pelvis, thus making the

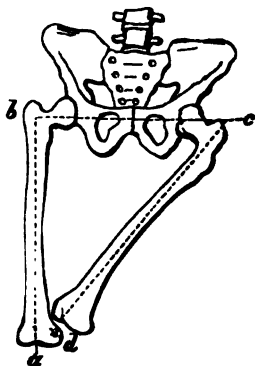


Fig. 3.

limb apparently shorter. He takes the position as in Fig. 2, only now the left hip is the diseased one, that is, the left hip is for this use of the diagram taken as being diseased.

I lay stress on this word "apparent" because by measuring with the tape you will not find those differences; indeed, the apparently lengthened, *i.e.*, abducted limb taken from any part of the crest of the ilium to the tuberosity of the tibia, or to the outer malleolus, measures absolutely shorter than the other, and *vice versa*. The only conditions that can produce real lengthening are excessive growth, fracture, separation of epiphyses or dislocation. I, therefore, advise you not to measure limbs suspected of hip disease with a tape. Do it rather thus: put the child flat on the back, place his body and lower limbs in as straight a line as possible. Do not, however, trust to eye alone; let him or a nurse hold one end of a string on the centre of the interclavicular notch, hold, or let some one hold (if assistance be insufficient, a weight hanging on the lower end of the string over the end of the bed answers equally) the other end, so that the cord passes between the feet; try to arrange the child so that the string passes over the xiphoid cartilage, the umbilicus, the centre of the pubes, and the space between the knees. Examine now the two inner malleoli. If they be not on a level the problem is solved. The child cannot tilt the pelvis without deflecting to one side some portion that ought to lie under the string. Therefore you may find it

impossible to get the child so that the cord shall fall on these median parts of the body—as you put one of them central another will deviate, and this will go on until you have to give up the attempt. The information you gain from this failure, if so it may be called, is as valuable as that which you can get from success, only remember that on whichever side of the string those central parts of the body lie, on that side is adduction of the limb, on the other is abduction, *i.e.*, shortening and lengthening respectively.

One other point about position deserves careful explanation. As the case goes on the hip becomes flexed; this is very often overlooked, indeed, I have often been called in to cases in which the attendant has believed the hip to be straight, because the child has lain with the limbs flat down on the bed; the error arises from not noticing the position of pelvis. For instance, this boy is so



Fig. 4.

lying, yet his hip is flexed, pass your hand behind the loin, and you feel a considerable interval between it and the bed. I take the diseased limb in my hand and gently raise it; now, on putting your hand under the loin you find it lying flat on the couch. Keep your hand there, and while I

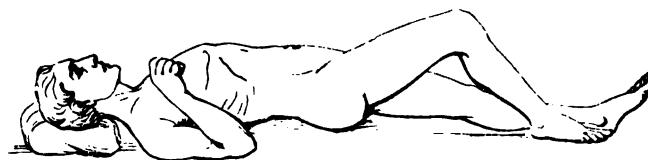


Fig. 5.

put the limb down again you will feel the pelvis lift, attain a somewhat more perpendicular position, while the loin again arches up, and once more there is a considerable interval between it and the bed. These two figures (6 & 7) show you the bones in those positions, and will help you to understand what I mean by saying that a child may lie with both limbs on the couch, and apparently quite straight, while one hip is in reality very much flexed.

I will now ask you to carry your mind back to

"fixity of the thigh," especially to remember that in the early stages of hip disease it is not due to

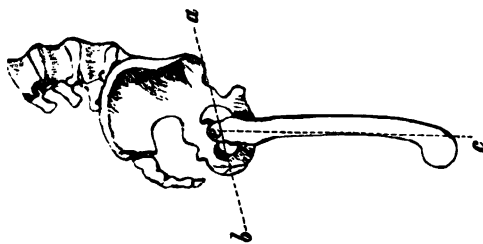


Fig. 6.

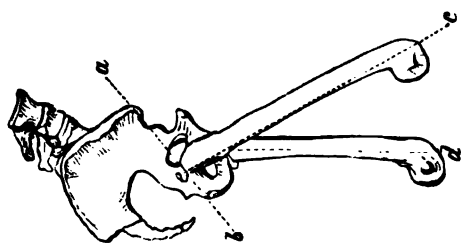
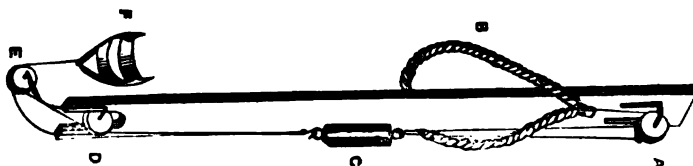


Fig. 7.

anchylosis, for it is entirely annulled by an anæsthetic; but that it is an effect of muscular action, reflex and emotional—that is to say, while the

tension, whose benefit lies in counteracting the morbid action of the long thigh muscles, and thus preventing that injurious pressure. The child in that third bed has extension applied by means of a weight. There are, to my mind, some great objections to this mode—chiefly, that the patient can move the trunk in any direction he likes, and therefore, though the legs themselves may be still, the body and pelvis move upon them; the hip is not immobilized. My extension splint, invented some years ago, obviates this. It consists of a long splint, provided with certain pulleys, as I now show you. To strapping plaister, so arranged as to form a loop under the foot, a cord is attached which plays over the lower pulley and passes *up* on the outside of the splint. To a perineal band, duly padded, which embraces splint as well as thigh, another cord passes round the upper pulley, and passes *down* on the outside of the splint. These two cords are connected by an india-rubber accumulator, which, when put on the stretch, drags the foot down; while the splint, when bandaged to both trunk and limb, immobilizes the hip.

This splint is most valuable in quite young



capsular muscles hold the head of the bone firmly against the acetabulum, the long muscles of the thigh—the rectus, the hamstring, and adductor muscles—force it (the head of the femur) with great power against the upper lip of the cavity. If you reflect that this abnormal pressure goes on night and day, you will expect some resultant evil. And when an opportunity occurs to examine anatomically a hip in not very early, but still early stage of disease, you will almost invariably find the cartilage of both femur and acetabulum ulcerated at their upper and slightly posterior aspect, the two ulcers being in contact. Later on, not merely the cartilage, but the bone, the upper lip of the cavity, is ulcerated; it is prolonged upward on to the dorsum ilii, while the *caput femoris* is on that aspect very much worn away,

This leads me to speak of treatment by ex-

children, or in all cases where the hip is considerably flexed. But when the patient is old enough, and when the limb is straight enough to allow of the use of a Thomas's splint, I strongly recommend its use. By its means you can let the patient get about and take exercise, without putting any weight on the entirely immobilized hip. Nearly all children with hip disease are strumous, or inclined to be so; a long stay in bed fosters that habit of body, and is injurious to health and sound nutrition. They are very inclined to tuberculosis of the mesenteric glands, and this further depraves nutrition. Exercise mitigates these evils, and helps recovery. I say, then, as soon as position of limb permits, get your patient out of bed and give air and exercise, taking care that the splint is well made, and that it really prevents movement of, and pressure on the joint.

THE
DIAGNOSIS AND TREATMENT
OF
GENERAL SEPTIC PERITONITIS.*

By C. B. LOCKWOOD, F.R.C.S. Eng.,

Surgeon to the Great Northern Hospital;
Assistant-Surgeon to St. Bartholomew's Hospital, etc.

SEPTIC peritonitis has hitherto been one of the most fatal diseases. Lately, however, some kinds of it have been found amenable to treatment by operation, although the disease was far advanced. It is reasonable to suppose that success would be greater if an earlier diagnosis were made, and prompt and determined treatment applied.

The object of this communication is to discuss, first, the question of diagnosis; and, secondly, the surgical treatment. Inasmuch as the subject is a very large one, I purpose to limit my remarks mainly to septic peritonitis, due to infection of the peritoneum from the abdominal viscera. This includes, of course, the various kinds of perforative peritonitis, and also those which are due to infection spreading through the fallopian tubes. The diagnosis of peritonitis due to injury or operation has not the same difficulties.

As my own experience of septic peritonitis with an internal source of infection is fairly extensive, I purpose to draw upon it rather than the writings of others. Moreover, no evidence will be adduced except that which is founded upon an actual inspection of the peritonitis. Mere clinical evidence is quite untrustworthy.

At the outset it is most desirable to have a clear comprehension that acute septic peritonitis includes many different kinds of disease. At one time it is caused by bacteria of comparatively slight virulence; at another, by bacteria of violent pathogenic properties. For instance, when the infection comes from the interior of the bowel, the colon bacillus and other intestinal bacteria are the main cause of the peritonitis. On the other hand, when the infection spreads from the fallopian tubes, the streptococcus pyogenes, the gonococcus, or other bacteria, are chiefly concerned.

* Read before the North London Medical and Chirurgical Society, March 12, 1896.

Now, it is acknowledged that all of these bacteria do not possess the same degree or kind of virulence. Moreover, as we shall presently see, they attack the peritoneum in very different ways. This diversity of virulence and of action is reflected in the clinical characters of the peritonitis which they excite, and also in its amenability to treatment. But, as yet, when confronted with peritonitis, we are still far from being able to say from the clinical symptoms that it is due to streptococci, to staphylococci, or to bacilli. Indeed, we cannot even say with any certainty whether a given case is one of peritonitis, or one of acute intestinal obstruction, due to mechanical causes. In many instances, this question can only be settled by an operation. The reason is not far to seek. In all the cases I have seen, the leading feature of acute general septic peritonitis has been acute intestinal obstruction.

In hospital practice, it is very sad to see the number of cases of acute intestinal obstruction in which delay has sealed the patient's fate. I cannot help thinking that this arises from the confused and involved way in which the symptoms of acute intestinal obstruction are taught and written about. In our latest works great stress is laid upon pain, vomiting, distension and facial expression, and little is said about the cardinal feature, which, obviously, is the non-passage of flatus or fæces. The symptoms which may accompany, such as pain, vomiting, distension, and so forth, are not peculiar to acute intestinal obstruction, but belong to other diseases as well. Clearly, they are of secondary value.

If I were asked the causes of acute intestinal obstruction, I should, relying on my own experience, at once divide them into inflammatory and mechanical. Paralytic obstruction is rare, and not, so far as I have seen, acute.

Now, in mechanical obstruction it is not difficult to understand how the cardinal symptom, the non-passage of fæces and of flatus,* arises. In acute septic peritonitis the reason is not so clear. I am accustomed to consider the problem from this point of view. It will be at once conceded that the intestines have two functions to fulfil; first, to

* I have but once met with a mechanical obstruction in which flatus was continuously passed. It was one of those very rare instances of partial enterocoele, a portion of the lumen of the gut being prolapsed through the femoral canal.

take part in the digestion and absorption of food ; and second, to propel their faecal and gaseous contents. Next, if any proposition in pathology is better substantiated than another, it is that which says that acutely inflamed structures cease to function. In acute septic peritonitis the whole thickness of the intestinal wall is not inflamed, only the serous coat. This, however, is enough to paralyse the muscular layers. Therefore, although I have been on the alert, and have watched carefully, I have never seen vermicular movements in septic peritonitis, nor can any be excited in the distended coils by tapping them. This absence of vermicular movements helps one to diagnose septic peritonitis, and also to discriminate inflammatory from mechanical obstruction. Indeed, in the earlier stages of mechanical obstruction, the vermicular movements may be increased in the attempts which the intestines make to overcome the obstruction ; but in the later stages they cease, because the muscular coat is exhausted by its efforts, or, because peritonitis has ensued.

The absence of vermicular movement in acute septic peritonitis helps to explain the non-passage of faeces or of flatus. I have hardly ever known any passage of faeces in acute septic peritonitis. In many cases the non-passage of flatus is absolute. In others, a little has been passed ; but so trifling in amount as not to invalidate the general rule. Occasionally, the administration of a turpentine enema evacuates a little flatus, but not enough to relieve. I am speaking of diffuse septic peritonitis. In many cases of localised septic peritonitis there is no complete intestinal obstruction. Thus, it must always be exceedingly difficult to diagnose intestinal obstruction due to mechanical causes from that which is due to inflammation. But a failure to do so cannot be blamed, or considered a fault. Our severest condemnation ought to be reserved for those who by not even recognising the existence of intestinal obstruction are guilty of irreparable delay. It is hard to comprehend how delay could occur if every one recognized the extreme gravity of the non-passage of flatus or faeces, with subsequent distension of the abdomen. Perhaps, as I have already said, the undue prominence given to more ambiguous symptoms may lead to misconceptions. Let us pass some of those symptoms in review.

First, as regards the general condition of the

patient. As a rule, those with acute septic peritonitis are exceedingly ill, but too much stress must not be laid on this. Not long ago, a youth with a gangrenous appendix and the accompanying septic peritonitis walked into the surgery at the Great Northern Hospital. Last year, a 'bus conductor walked about and visited chemists and doctors whilst suffering from undoubted septic peritonitis, due to a perforating duodenal ulcer. The last case had had obvious acute intestinal obstruction for some hours, and had, as is lamentably frequent, been treated with purgatives.

Next, the voice of those who are on the brink of the grave from septic peritonitis may be loud and even resonant. A young lady who had been operated upon for perforating gastric ulcer, smiled, and said she felt better with almost the ordinary intonation of her voice ; and yet she was dead in a few hours. That she should say she felt better is quite usual. Recently, another patient dying of septic peritonitis made the same statement, and seemed very sanguine, but succumbed in a few hours. This mental state seems to mislead those who listen with credulity to the statements of patients. The doom of both of those whom I have just mentioned was foretold by the pulse. In each it was almost of uncountable rapidity.

Much has been made of the expression of the face in septic peritonitis. I have heard it called by various names, such as *facies abdominalis*, and *facies hippocratica*. It is unnecessary to repeat the classical descriptions. It is the face of a dying person, obviously it is not a sign to be waited for. Much ought to have been done before it is seen.

The temperature is most misleading in septic peritonitis. In nearly every case in which I have operated the temperature has not been raised, or has been subnormal. In January, 1896, I operated upon a case of gangrenous appendicitis, in which the temperature was 102° F. This fact, taken with symptoms of acute intestinal obstruction, and the co-existence of fluid within the pelvic peritoneum, helped the diagnosis. The absence of temperature would have weighed but little. For pyogenic bacteria produce ptomaines which cause elevation of temperature, but it is obviously unreasonable to suppose that all bacterial poisons are the same. On the contrary, it is more reasonable to infer that some may depress the body temperature.

In septic peritonitis the pulse affords much more

important indications than the temperature. After abdominal operations, it is more important to watch the pulse than the temperature, and the same holds good in any suspected case of peritonitis. I should feel incredulous if I heard of a case of septic peritonitis without an acceleration of the heart's action. A pulse rate over 100 in an abdominal case is a sign of some import, especially if the rate grows quicker. In the later stages the pulse gradually increases in rapidity, until at the end it is uncountable—a mere thread. Whether this acceleration is due to a reflex nervous action in the heart, to ptomaines, or to an infection of the substance of the heart with bacteria I do not know. The last point might easily be ascertained, and I regret not to have had the time to pursue the investigation. The rapid pulse is accompanied with great coldness of the extremities. I have not systematically noted the pulse in cases of mechanical obstruction; but in some my notes say that it beat 90 per minute, and was of full volume.

The intestinal obstruction of acute septic peritonitis is almost always accompanied with vomiting. The latter is therefore a confirmatory symptom. But too much must not be inferred from its frequency, or even from its absence. Recently, I operated upon a case in which vomiting was quite a minor feature, although she had a pulse of 130, and was almost moribund. Not long ago a case of vomiting, with violent abdominal pain and constipation, was mistaken for acute intestinal obstruction, probably due to peritonitis. The man had been taking a mixture of sandal oil and copaiba. Sometimes, the absence of vomiting in septic peritonitis is due to the administration of opium. In the cases I have seen, the vomiting of peritonitis has remained bilious for much longer than it does in mechanical obstruction. This abdominal distension following upon the presence of flatus or of fæces, is of great diagnostic value; it is analogous to the distended bladder in retention of urine, or the distended gall-bladder in biliary obstruction. A distended abdomen is a grave complication, and indicates that the peritonitis is much advanced; it also renders an operation more difficult, and much more dangerous. Sometimes it is possible to ascertain by physical examination that both the large and small intestine are full of gas. In acute intestinal obstruction, due to mechanical causes acting upon the small intestine, the bowel below the constriction

is empty, or, at the most contains a little gas. A mechanical obstruction in the large intestine, accompanied with acute symptoms is most unusual. But the chronic intestinal obstruction which complicates growths in the large intestine sometimes becomes acute towards the end; this, however, could hardly mislead.

Now and then the inflammation of the peritoneum spreads to the abdominal wall, and it becomes cedematous, and sometimes red. I have only observed this in very advanced cases, and especially when there has been at first a localised septic peritonitis about the appendix.

In acute septic peritonitis a rectal examination often yields important information. It is strange to observe that this most valuable aid to diagnosis is very often omitted; but great caution must be exercised in drawing inferences from what is felt. For instance, a woman with acute intestinal obstruction had a mass high up the right side of the rectum. This felt like an inflamed vermiform appendix, with surrounding peritonitis. As a matter of fact, it was a mass of intestines matted together by inflammation around the right fallopian tube. Doubtless the previous history of attacks of inflammation helped to mislead.

In acute septic peritonitis collections of fluid in Douglas's, or in the recto-vesical pouch, can very often be felt by rectal examination. Twice in one week I met with collections of fluid in the recto-vesical pouch, which felt like a distended urinary bladder. In one, the fluid swelling could not be felt above the pubes. At the operation, acute septic peritonitis was found with much fluid in the pelvis. The patient, who was a middle aged man, seemed to bear the operation well, but died with great suddenness a few hours after. The cause of the peritonitis was never discovered, as an examination was not allowed. In the other case, the fluid swelling could be felt like a distended bladder above the pubes, but projecting towards the left linea semilunaris. Fluctuation was very free betwixt the finger in the rectum and the hand upon the abdomen. This swelling also contained gas which had been made by colon bacillus, of which the fluid was full. In this case, the pelvic swelling was no clue whatever to the cause of the peritonitis. The latter was due to a perforating ulcer of the duodenum. I never suspected this, because, at the operation the upper regions of the peritoneum

seemed not to be inflamed. At the examination, the peritoneum might have been divided into three zones; an upper, in which was much peritonitis; a lower, which was the same; and a middle, in which hardly any peritonitis could be seen. We found nothing to show how the upper zone had communicated with the lower. Except for the collection of fluid in the pelvis, this man's symptoms were the same as those of an acute mechanical obstruction. I have often discovered fluid in the pelvic part of the peritoneal sac in septic peritonitis, and I believe that a rectal examination would show that it is to be found in at least half the cases. It should always be looked for, and when the fluid is present, the rectum feels very hot, and pressure with the finger is exquisitely painful.

I have already said that the absence of pain in the latter stages of septic peritonitis may mislead. In 1893, I operated upon a girl with diffuse septic peritonitis, who had no pain, and free abdominal respiration. But the presence of pain seems seldom to afford reliable assistance. Now and then it is definitely localised at the beginning of the attack. Twice, in perforating ulcers of the duodenum it was very severe, and localised beneath the right costal margin; in ulceration of the vermiform appendix it was intense over the iliac fossa. But I have just mentioned a case of perforating ulcer of the duodenum, in which there was absolutely nothing which pointed to either the perforation, or led me to suspect the peritonitis which it caused beneath the liver. Recently, I helped a colleague to operate upon a case of gangrenous appendicitis. The patient's abdomen was distended and rather tender, but nothing whatever pointed to a serious and fatal lesion of the vermiform appendix. I have operated upon exactly similar cases myself during the last two years.

Patients sometimes speak of a burning pain in the whole abdomen, and now and then the slightest pressure of the finger causes extreme pain. But sometimes pain is absent, as in the case of a girl who not only had no pain, but also breathed with the abdomen, although she was dying of general septic peritonitis. She had, however, complete intestinal obstruction.

Amongst the other general symptoms of acute septic peritonitis I need hardly allude to the thoracic respiration, or to the decubitus, or to the rigidity of the abdomen. All are indications to

be taken into account, but no one would like to base a diagnosis upon their presence or absence. I have more than once seen abdominal breathing and outstretched legs in quite advanced septic peritonitis.

The diagnosis of septic peritonitis is sometimes rendered easier by a knowledge of the previous history. For instance, a gastric ulcer, gall-stones, appendicitis, genito-urinary diseases, the passage of uterine sounds, and so forth, may be shown. As a rule, however, the previous history is misleading. The foregoing is but a passing glance at the clinical symptoms of septic peritonitis as they present themselves to my mind. The diagnosis is obviously surrounded by great difficulties. The symptoms which are the most significant and trustworthy are (1) The acute intestinal obstruction. (2) The abdominal distension. (3) The absence of vermicular movements. (4) The collection of fluid in the pelvis. (5) Evidences of inflammation about the cæcum, fallopian tubes, gall bladder, or elsewhere. (6) The pulse, (7) and last, the vomiting and other symptoms are to be taken into account, and may afford confirmatory evidence.

As I have already said, the differential diagnosis betwixt acute septic peritonitis and acute mechanical obstruction is always difficult, and oftentimes impossible, without an operation. But perhaps the following slight differences may help a differentiation. (1) The intestinal obstruction is more often absolute in mechanical obstruction. (2) The vermicular movements persist for some time, and may be increased in mechanical obstruction. (3) The pulse has a good volume, and is not markedly accelerated in mechanical obstruction. (4) Fluid does not collect in the pelvis in mechanical obstruction, and evidences of inflammation of the appendix, fallopian tubes, gall bladder, and so forth, are wanting. (5) Elevations of temperature are less common in mechanical obstruction. (6) Fæcal vomiting occurs earlier in acute mechanical obstruction.

Laparotomy may have to be done as a final step in the diagnosis. An exploratory laparotomy is a confession of impotence. A diagnosis ought to precede an operation, and not follow it. The need for an accurate diagnosis is clearly shown by a case which has been mentioned. In it the abdomen was opened below, and a peritonitis in

upper part completely overlooked. The success of an operation depends entirely upon the precision and rapidity with which it is done. Neither of these is possible without at least an approximate diagnosis. But it is obvious that the treatment of diffuse septic peritonitis must ultimately depend upon its causation. In performing laparotomy for septic peritonitis during the past two years I have found gangrene of the vermiform appendix, ulceration of the vermiform appendix with faecal concretions in the peritoneal cavity, pyosalpinx, salpingitis, perforation of the ileum, and perforation of the duodenum. In one case the appendix was known to be at fault; several others were known to have general septic peritonitis, but its starting-point in the gangrenous appendix, in the septic fallopian tube, or duodenal ulcer, was quite unknown. In one case peritonitis was diagnosed in mistake for an acute mechanical obstruction, and in another acute mechanical obstruction was diagnosed in mistake for peritonitis. In all the symptom which called most imperatively for relief was the acute intestinal obstruction. Now, whether this is due to mechanical or inflammatory causes delay is most irrational. Who would think of leaving a gangrenous appendix, a faecal concretion, or intestinal contents in the peritoneal sac? or, how could a mechanical obstruction be relied upon to relieve itself? My own experience leads me to believe that in acute intestinal obstruction the dangers of laparotomy are trifling in comparison with those of delay. But by laparotomy I do not mean those groping operations which aim at nothing and end in disaster. Success can sometimes be obtained in the most desperate cases* by measures taken before, during, and after the operation. It is to be deplored that surgeons are usually called upon to treat septic peritonitis when it is far advanced. Owing to want of food, constant vomiting, pain, sleeplessness, and mental distress, the patients are in an exhausted or critical state. Therefore, it is necessary, before the operation to temporarily improve their condition by subcutaneous injections of strychnine and with enemata of brandy and hot water. During the operation the room is kept at a temperature of 70° F. and the patient is

put upon a hot water bed and kept well covered, the extremities being wrapped in wool or flannel. If a hot bed is not at hand hot water bottles can usually be obtained. All the sponges and instruments should be kept warm at a temperature of 105° F. The anæsthetic should be given for as short a time and as sparingly as possible. When the general condition and the pulse are both at their worst a temporary improvement nearly always ensues during anæsthesia. I have never yet known a case of general septic peritonitis succumb during the operation.

I do not now propose to discuss at length the operation. Much depends upon the diagnosis. But hap-hazard proceedings undertaken without a definite plan are much to be condemned. As a rule the abdomen is opened by an incision in the middle line or above the umbilicus. When the abdomen is opened below the umbilicus, and nothing is immediately seen, the vermiform appendix and cæcum should be forthwith explored and actually seen. Ulceration, gangrene, or other inflammatory troubles about the appendix are so common that this step is often crowned with success. Moreover, the condition of the cæcum is a guide to the seat of the obstruction. In inflammatory obstruction it is distended together with the rest of the large intestine. If nothing be found in the right iliac fossa the pelvis and fallopian tubes are examined. Perforated and collapsed intestine very often falls into the pelvis, and in women the fallopian tubes are frequent sources of infection. Should nothing abnormal be discovered in the lower part of the abdomen, the question at once arises as to whether the stomach and duodenum should be explored. After the case in which, misled by the fluid in the pelvis, a duodenal ulcer was overlooked, I should explore the upper part of the abdomen through a separate incision above the umbilicus. This would be a very small addition to the danger or shock of the operation. The intestinal distension is a dangerous and embarrassing complication of operations for general septic peritonitis. The tense intestines are greatly in the way, and their coats are apt to tear during the exploration. Moreover, it is essential to success to adequately relieve this distension. In a number of instances I have evacuated the intestinal gases with a fine trocar and cannula, and emptied off their faecal contents

* "The Surgical Treatment of Diffuse Septic Peritonitis, with Successful Cases," by C. B. Lockwood. Med. Chir. Trans. 1895, vol. 78, p. 1.

through small longitudinal incisions, which are afterwards closed with sutures. If a puncture leaks it is converted into an incision so as to drain better, and make sure that the distension is relieved.

The last step of the operation is to wash out the abdomen methodically with several gallons of hot water, 105 to 110° F. The patient needs careful watching at this stage as the hot water seems a little uncertain in the action upon the heart. It is unnecessary and harmful to take any steps to sponge out the fluid. It is sucked out through a large glass drainage tube which is placed in the pelvis. The wound is closed and dressed in the usual way.

When the operation is over a determined struggle begins to prevent the patient dying of shock. Hypodermic injections of strychnine and ether are valuable, but especially heat and rectal injections of brandy and hot water. Feeding by the mouth is undertaken with great caution. Now and then patients do not vomit what is given by the mouth, but the risk is great. Therefore, well peptonised nutrient enemata of milk, meat essence, and brandy are relied upon. Thirst may be relieved by copious enemata of warm water. During, at least, the first twenty-four hours a medical man should be in constant attendance upon the patient.

Since November, 1893,* I have operated upon ten cases of diffuse, or general septic peritonitis. Of these cases three recovered, and seven died. In considering these recoveries, and the heavy mortality, the inevitably fatal character of the disease ought to be remembered. Of the cases which recovered, the peritonitis originated in the vermiform appendix, in the fallopian tube, and in a perforation of the ileum.

Of the patients who died, one had gangrene of the vermiform appendix; one had ulceration of the vermiform appendix; two had perforations of the duodenum; one is thought to have had a tubercular ulceration, and perforation of the intestine; one had septic endo-metritis and pyosalpinx; and in one the cause was quite unknown.

In some of these cases there had been lamentable delay. In a case with most urgent symptoms, forty-eight hours had been allowed to elapse; and

* I begin at November, 1893, because my notes begin then, but I cannot recall a successful case before that.

in an appendix case five days had been wasted upon drugs and palliatives. But I do not wish to convey the impression that all cases of septic peritonitis could recover if an operation was done soon enough. For instance, here is a drawing of the peritoneum from a case of diffuse septic peritonitis. It is evidently due to a bacillary invasion of the peritoneum, and the clinical history shows that the bacilli almost certainly originated from the interior of the bowel.* In this case the bacteria are abundant in the lymph upon the surface of the serous membrane, but they have not penetrated its depths, which seem comparatively healthy. Indeed, it looks as though the surface of the peritoneum might be freed of its parasites, and that the depths had undergone no change which forbade recovery. Further, the bacteria are probably the colon bacillus, which belongs to a species which has not, as a rule, very strong pathogenic properties.

Now let us glance at another case of general septic peritonitis. In it the lymph upon the surface of the serous membrane is covered with strings of micrococci; and, in addition, the bacteria have invaded the substance of the peritoneum, and penetrated as far as the subperitoneal fat. This constitutes a true erysipelas, or even a cellulitis, of the peritoneum. None of the means which we at present possess could deal with such an invasion as this. Moreover, the streptococcus of erysipelas or of suppuration is highly virulent in other respects, and is much more to be feared than the colon bacillus. Clearly, a streptococcus peritonitis such as this must, with our present knowledge, be more fatal than bacillary peritonitis. In streptococcus peritonitis we may have something to hope from serum therapy, which as yet is only in its infancy. Other kinds of peritonitis might also be mentioned, about which singularly little is known, such, for example, as staphylococcus peritonitis, and pneumococcus peritonitis. Each of these must have peculiarities of their own.

In bringing this communication to a conclusion, perhaps it may be well to recapitulate the theses maintained in it. First, that in nearly every case the clinical symptoms of general septic peritonitis

* This and the next case are given at length in the author's Hunterian Lectures on Traumatic Infection, London, 1896, p. 12, etc.

are those of acute intestinal obstruction. Second, that in many cases general septic peritonitis cannot be differentiated from acute mechanical obstruction. Third, that to prevent a mechanical obstruction, a gangrenous or ulcerated appendix, a septic fallopian tube, or a perforation of the alimentary tract being overlooked, an early laparotomy ought to be performed. Fourth, that after operation even the most desperate cases may recover.

EARLY GENERAL PARALYSIS OF THE INSANE.*

BY

J. S. RISIEN RUSSELL, M.D., M.R.C.P.,

Assistant-Physician to the Metropolitan Hospital, and
Pathologist to the National Hospital for the Paralysed
and Epileptic, Queen's Square.

GENTLEMEN,—The two cases to which I propose to call your attention are instances of what I regard, on clinical evidence, as early general paralysis of the insane. I have selected these two cases because they serve to invite discussion, more especially on three important points, in this interesting and controversial subject. The first of these points is the difficulty of diagnosing between some cases of early general paralysis and cases of cerebral syphilis; the second point is the part played by syphilis in the etiology of general paralysis; and the third, the value of grandiose delusions as a symptom of early general paralysis.

The clinical histories of the two cases are, briefly, as follows:—

Case 1. A. B., æt. 33 years. Married 12 years. Healthy family of six children. Wife no miscarriages, but one child died 17 hours after birth, and another 7 weeks old. The patient's father died suddenly. No history of nervous disease or insanity in family. Patient had previously enjoyed good health, with the exception of having had syphilis sixteen years ago. No history of alcoholic excess.

His present illness began with an epileptic fit

twelve months ago. He has had two "sensations" since, but no actual fit with convulsions or loss of consciousness. His friends have only noticed alteration in character of his speech during the last few months, and he has been losing strength and sexual power during a like period. There has never been any exalted ideas. He is a fairly nourished man, and complains of nothing but feeling weak. He is emotional, and his speech presents the typical characters of that which we are in the habit of associating with general paralysis. The lines and wrinkles on his forehead are well marked. He is able to perform all facial movements, the lower being attended with considerable tremor. There is coarse tremor of the tongue on protrusion. There is no ptosis or squint, and the pupils are equal and react normally. His grasps are feeble, and his gait a little uncertain, though there is no marked defect in the latter. His tendon reflexes are all increased. There is no loss of muscular sense. He is able to stand with feet together and eyes closed without becoming giddy. There is no blunting of cutaneous sensibility. The optic discs are slightly high-coloured, but the margins are well defined, and there is nothing otherwise abnormal in the fundus.

Case 2. C. D., æt. 47 years. Married 3 years. No children. Nothing worthy of note in his family history. He enjoyed good health previously, with the exception of a history of a rash and sore throat about four years ago. His wife knew him at that time, and says he had sore throat, but no rash. Patient is not aware of having had primary specific lesion. He has always been temperate as regards alcohol. His wife first began to notice "childishness" at the end of last year: but there has been gradual failure of sexual power for quite a year, and the patient states that he has been losing strength for about a year, in spite of his appetite being greatly increased. His wife states that he eats far more than he used to. Two years ago he failed in business, and this was preceded and followed by a great deal of mental worry and anxiety.

The patient is a man of slight build, is poorly nourished, and complains of losing strength. There is marked dementia; he loses his way, goes into other houses in mistake for his own, and does various silly things. He has never showed the

* Notes of a Paper read at the Metropolitan Hospital.

slightest sign of exaltation, and no such evidence can be obtained by direct or indirect questions. His speech is characteristic of general paralysis. The forehead is smooth, and attempts to raise the eyebrows or frown result in next to no movement. The lower facial movements can be carried out, and that with little tremor. The tongue on protrusion shows slight fine fibrillary tremor. There is no ptosis or squint. His pupils are equal, and react normally. His grasps are feeble, and there is evidence of general muscular enfeeblement. All the tendon reflexes are increased. There is some loss of muscular sense in the right arm. Romberg's sign is absent. Cutaneous sensibility seems blunted generally, but more so on the right extremities.

Comparison of the Cases.

If we now compare these two cases we find that in neither is there, or has there been, any exalted ideas, and in neither is there inequality of pupils or failure on the part of the pupils to act to light or on accommodation. Both present evidences of motor weakness in both the tendon reflexes are increased, and in both there is failure of sexual power.

Thus far the cases agree, but they differ markedly in many respects. In the first patient there is a distinct history of syphilis, whereas, in the second case, only a doubtful history can be obtained. No exciting cause other than syphilis can be assigned in the first patient, but mental worry and anxiety connected with failure in business preceded the development of symptoms in the second case. An epileptic fit is the first evidence of brain disturbance that we can obtain in the first case, while progressive dementia characterised the onset of the disease in the second case. As so often happens when dementia is a marked feature of the early stages of the disease, tremors of the tongue and face are comparatively slight in this patient. The first patient is subject to depression and is emotional, whereas, the second, though the subject of marked dementia, is not at all emotional. The difference of facies in the two cases is striking, the wrinkled forehead in Case 1 giving rise to a fixed frown, as contrasted with the smooth forehead and vacant expression in Case 2. The former patient is able to perform all voluntary movements of the facial

muscles, including the acts of raising the eyebrows and frowning, whereas, in the latter case, though the lower facial movements can all be carried out, there is little result when he attempts to raise the eyebrows or to frown. Speech, though defective in both cases, is much more characteristically so in Case 1. Cutaneous sensibility and muscular sense are both intact in the first patient, but in the second, cutaneous sensibility does not appear to be very acute anywhere, but the defect is more noticeable in the right limbs than in the left, and there is some loss of sense of position in the right superior extremity.

Difficulties of Diagnosis between General Paralysis of the Insane and Cerebral Syphilis.

As I said in my opening remarks, I regard both these cases as instances of general paralysis, though they are different types of this affection. The chief difficulty with which we are confronted in arriving at a diagnosis, more especially in the first case, is as to the possibility of cerebral syphilis, in spite of the long interval between the primary lesion and the onset of the symptoms.

It has always been admitted that the diagnostic problem is often an exceedingly difficult one where we have to distinguish clinically between cases of cerebral syphilis and early general paralysis of the insane; but of late the difficulties have been multiplied in that there are those who hold that many of the cases we formerly regarded as general paralysis ought, in reality, to be classed with certain other cerebral disorders, and that no mean proportion of them are instances of cerebral syphilis.

We are, I think, justified in looking for some of the symptoms which more especially characterise cerebral syphilis, e.g., early paralysis of cranial nerves, localised motor or sensory paralysis, optic neuritis, insomnia, the peculiar nocturnal headache, and so on, in addition to the history of syphilitic infection, before diagnosing cerebral syphilis rather than general paralysis in a case whose characters conform to the clinical picture we are in the habit of associating with the latter disease. On these grounds, therefore, I think that all the clinical evidence in the first case points to the disease being general paralysis of the insane.

The history of syphilis in the second case is too doubtful to justify any serious question of the

existence of cerebral syphilis being based on this point, nor are there any of the coarser manifestations of syphilis, or the special features of the disease already alluded to, to warrant such a diagnosis. The case, however, has one feature which is common in cerebral syphilis as in one class of general paralysis, viz., dementia, as the chief symptom; but in the absence of some more characteristic signs of cerebral syphilis, I see no reason to look on the case as other than one of general paralysis of the insane.

Certain as the diagnosis appears to me to be in these two cases, I have, nevertheless, to admit that some cases of cerebral syphilis so closely simulate what we are in the habit of regarding, on clinical grounds, as general paralysis, that every test should be applied before an absolute diagnosis is made. It is, I think, our duty, in every case of supposed general paralysis, to subject the patient to the influence of rigorous antisyphilitic treatment for a reasonable time before relegating him or her to a class of cases which we regard as beyond our power to aid by drugs. Accordingly, I have put both these cases on a course of mercury and iodide of potassium. The second case has not been under the treatment long enough (ten days) to justify any conclusion at present; the first case, however, has been under treatment for three weeks, and his gums are now affected; but up to the present I cannot say that there has been any noticeable improvement. Ten days ago he seemed brighter, and his speech appeared slightly improved; but when seen four days ago, the condition was much as when the treatment was first commenced.

Syphilis as an Etiological Factor in General Paralysis.

The question whether syphilis does or does not produce general paralysis is a much-contested one; there are those who hold that it does so, while there are other authorities whose opinions have been equally strongly expressed to the contrary.

In the absence of any other assignable cause, it is difficult to escape from the belief that syphilis may possibly stand in direct causal relationship to the first case, so that if we admit that the case is one of general paralysis, it is one which may be claimed to support the view of those who hold that syphilis plays a part in the causation of this disease.

Grandiose Delusions.

I have been much struck by the infrequency of occurrence of grandiose delusions as an early indication of general paralysis, as seen in cases in the out-patient rooms of this and other hospitals. The inflated, loquacious, self-satisfied man, with unbounded wealth, or unprecedented strength, and so on, is conspicuous by his absence. The patients usually give no indication of having any exalted notions with regard to health, physical powers, and so on; on the contrary, the majority of them tell us that they feel that they are gradually losing strength, a state of things that is abundantly manifest on physical examination.

So much stress has been laid on the extravagant delusions and emotional exaltation as early indications of general paralysis by many writers, that the absence of these symptoms has often been responsible for failure to recognise otherwise typical instances of the disease. It must be remembered in this connection that many cases of general paralysis run their whole course without any such grandiose delusions, and that when these do occur, they rarely make themselves evident until some considerable time after a skilled observer has been able to detect many other evidences of the disease.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Reported by J. T. CONNER, M.D.

Sebaceous Tumours of the Scalp assuming Malignancy.

A woman, æt. 73, sent by Mr. Treacher Collins, had a conglomerate mass of tumours on the back of the head, firmly adherent to the bone, infiltrating and destroying the scalp. The tumours were spherical, and varied in size from a pea to a large plum. The smaller ones were hard, but the larger soft and fluctuating. The condition began ten years ago as "a smooth yellowish-white lump."

Mr. Hutchinson said that the disease evidently began as an ordinary sebaceous cyst which had as-

sumed malignant properties, reproduced others, and invaded the scalp. The transformation of benign into malignant growths was now a well-recognised fact. He had published several cases in which sebaceous tumours of the scalp had become malignant and even produced secondary growths all over the body. A portrait was shown, in which the secondary growths not only covered the scalp, but were distributed generally in the skin.

Persisting Œdema of the Eyelids from retention of lymph in spaces which communicated by dilated lymphatics with a similar space behind the ear.

A boy, æt. 8, brought by Dr. Herbert, showed marked swelling of the right eyelids, which were soft, puffy, and slightly bluish. Pressure on either lid caused the swelling to disappear in that one, whilst increasing that in the other. Pressure on both removed the swelling entirely from the eyelids, but caused a similar but slightly lobulated swelling to appear behind the right ear. Thus the fluid could with the greatest ease be driven from place to place. At the same time a number of cords could be seen to appear beneath the skin, running from the eyelids to the swelling behind the ear. Dr. Herbert had counted six.

The affection began two years ago, when the swelling of the eyelids occurred without obvious cause. Five months ago an attack of facial erysipelas occurred. Since this the swelling of the eyelids has been increased, and the swelling behind the ear appeared. His general health has always been good.

Mr. Hutchinson said that the case was quite unique in his experience. There was evidently a collection of fluid in dilated lymph spaces in the eyelids and behind the ear, the former communicating with the latter by dilated lymphatic vessels. No explanation of the causation was forthcoming. The attack of erysipelas had occurred since the onset of the disease. Moreover, this, even when recurring, was not known to produce anything more than persisting solid œdema.

Eruption from the use of Arsenic.

The patient was a boy, æt. 14, sent by Dr. Crozier. He had been attending a general

hospital for some months for an attack of chorea, without benefit. Two months ago he came under the care of Dr. Crozier, who prescribed ℞^{xv} of liq. arsenicalis three times a day. In two weeks he was nearly well. He then left off the medicine for nine days, but resumed it again in the same doses, and has continued it up till now. The chorea is now quite cured.

Four weeks ago he noticed that his neck was red and scaly, two weeks later the arms became similarly affected, first, at the flexures of the elbows. A week later the legs were attacked.

Now, the skin is brown and earthy all over, as well as rough and in a goose-skin condition. The hands are congested, dusky, and desquamating. The elbows are congested both on the flexor and extensor surface. The latter is covered with scales like the psoriasis patch, but duller, and not of such silvery whiteness. The popliteal spaces are erythematous. The hair follicles of the extremities are enlarged and prominent, in the condition described by Tilbury Fox as "cacotrophia folliculorum." The patient complained of aching in the legs, but there was no numbness. Mr. Hutchinson pointed out that though there was no evidence of neuritis the aching of the legs was a symptom of arsenical poisoning. The tendency of the arsenical eruption to imitate psoriasis was pointed out. Portraits demonstrating this, as well as the keratoses of the palms and soles, and the epitheliomata produced by the prolonged use of the drug, were shown. One feature in the present case was that the conditions had been produced not by long-continued use of the drug, but by full doses given for a short time.

Myositis Ossificans in a Child, confined to the Pronatores Radiorum Teretes.

The patient was a boy, æt. 6. The left forearm was completely fixed in the position of pronation, but the movements of the elbow-joint were free. A hard band, like a bony ridge, could be felt running obliquely from the inner condyle to the radius, in the position of the pronator radii teres. On the right side a similar but less advanced condition was present. He could partially supinate on this side. The disease commenced when he was two years old. Stiffness and difficulty of supination in the left arm was first noticed. This

gradually increased, and the movement was quite lost in about a year. The right arm was noticed becoming affected a year and a half ago. It is gradually getting worse. Mr. Hutchinson considered it remarkable that the disease, which was now of four years duration, should have remained confined to one pair of muscles. He knew of no similar case. He thought that excision of the affected muscles might prove of benefit.

FATAL CASE OF HÆMORRHAGE UNDER ANÆSTHESIA.

BY

CHARLES CARTER BRAINE, F.R.C.S.

A DEATH from hæmorrhage whilst under an anæsthetic is of extreme interest, chiefly on account of its rarity. The following notes, which were written immediately after the occurrence, may be taken as an accurate report of the case.

A powerfully-built man was going down into a cellar, when the trap-door fell upon him, striking him in the pit of the stomach, and jamming him against the side of the opening. This occurred at 11 a.m., and he was brought to the hospital in a state of collapse; pulse feeble (120), pupils dilated, legs drawn up, and slight movement of abdomen during respiration. Skin cold and clammy. At 3 the pulse was weaker, and had increased to 140; and at 3.30 it was decided to operate. The anæsthetic—ether—was commenced at 3.40 with the Ormsby inhaler; the patient became anæsthetised very rapidly, and was taken into the theatre. Upon performing laparotomy, the peritoneal cavity was found to be full of blood and clots, the removal of which was commenced by flushing out the abdomen with warm water—but, curiously enough, no bleeding point could be found. It was then considered to be a case of ruptured liver, and this organ, together with the stomach, were carefully searched for a lesion, but with a negative result. The blow was said to have been received “in the pit of the stomach,” and hence this part was carefully examined, and much valuable time lost thereby.

The patient had now been under ether between twenty to thirty minutes, when, quite suddenly,

there was a gush of blood welling up everywhere amongst the intestines, and upon baling this out two or three large branches of the superior mesenteric were found actually pumping. Bystanders remarked that the hæmorrhage was more free than in any case of post-partum they had seen. Now this is a very interesting point, for these vessels could not have been bleeding all this time, or the patient would certainly have been dead ere this; and I presume that this hæmorrhage must have been due to recovery from the shock of the injury through the stimulating action of the ether, and the flushing of the peritoneum. The patient was now in a very critical condition, with dilated pupils, half-closed eyelids, radial pulse hardly perceptible, and beating at the rate of 140 per minute; and no pulsation to be felt in the temporal or facial arteries.

One quart of saline solution was now introduced by siphon action into the left median basilic vein—this very soon had a marked action for the better, and the pulse could easily be counted at the wrist. The anæsthesia kept up was now of the lightest.

The presence of a large rent in the mesentery was now discovered, together with a ruptured small intestine, and so matters became much more serious than before. Quite a foot of the intestine was resected; the ether apparatus was now changed for Rendle's mask, as I wished the patient to get plenty of fresh air with the ether, and I think the change was beneficial. He now began to lose ground again, the pulse had become a running one, and a brandy enema was administered, and hot cloths applied to the precordial region and nape of the neck.

To my horror and the utter despair of the surgeon, another rent in the intestine was found, and another anastomosis had to be performed, and this I consider was the last straw. When this had been effected, the toilet of the peritoneum attended to, and the sutures all *in situ*, the time was 6 p.m., or two hours and twenty minutes from the commencement, and no ether had been inhaled for the last fifteen minutes. The patient was now extremely bad, pulseless and cold, pupils widely dilated, and respiration feeble, which up to the present time had not given the least anxiety. The legs had been elevated from the very commencement, but they were now slung to a stretcher pole,

and fixed almost vertically—the brachials and femorals compressed, and the vapour of ammonia applied to the nostrils. The ammonia certainly stimulated respiratory efforts for a time. It was very evident that he could not be moved out of the theatre, and as he was sinking fast, his friends were admitted for a few moments to see him. Respiration had almost ceased. I now noticed that his pupils had contracted, and my hopes immediately revived—the friends were turned out, artificial respiration performed, ammonia held to the nostrils—a little brandy poured down his throat, and the hot cloths resumed; more saline solution was also injected, making in all two quarts; oxygen was also inhaled, being applied for four or five inspirations at a time during the artificial respiration; air entered freely, and the pulse could be felt in the carotid and femoral arteries; and after about fifteen minutes of this treatment he began to breathe on his own account, deglutition took place, and the conjunctival reflex was present. Oxygen was given at intervals, as it seemed that if we could only get oxygen to his centres, he might possibly recover.

The pulse was now good, and breathing regular—phonation was also present. Everything now appeared so satisfactory, that the resident officer undertook to sit up through the night and give oxygen, and perform artificial respiration, if necessary; but at 6.40 breathing gradually ceased, the pupils again became widely dilated, conjunctiva insensitive, and artificial respiration and the administration of oxygen were of no further avail.

Post mortem.—No other lesion was discovered. All the viscera were healthy, the colour of blood and tissues quite normal.

This case illustrates the great value of intravenous injection of saline fluid in cases of severe hæmorrhage: the two other cases in which I have seen it used both recovered; they would certainly have died on the table, had it not been exhibited. The ammonia had a marked effect upon the pulse and respiration, but of short duration. The administration of oxygen during the artificial respiration was also of value. The sudden change in the size of the pupil from one widely dilated to about the normal, when we had given the man up as dying, was very curious, and led to the re-establishment of respiration, and the prolongation of life for another forty minutes.

THERAPEUTICAL NOTES.

Thioform as an Aseptic.—Dr. A. Steuer has been using this material in several surgical ulcerative conditions, with results, in his opinion, superior to those obtained by iodoform or carbolic acid. Thus, varicose ulcer of leg, soft venereal sores, weeping eczema, purulent otitis media, have all yielded to this medicament. He concludes his article by recommending thioform in all cases where profuse suppuration is present, or where any secretion exists that requires to be dried up. It is non-poisonous, as dear as iodoform, but, being lighter as a powder, its application is cheaper. (*Wien. Medic. Woch.*)

Tachycardia.—E. Hausler reports a case of the following nature:—The patient suffered from muscular rheumatism; this was followed by sudden onset of tachycardia, with a pulse rate of 200. Antirheumatics and digitalis proved utterly useless. Quinine was then given in large doses (30 grains in divided doses within two hours), with a most brilliant result—within three or four hours the pulse sank to 80. For two years the patient remained well, and then a similar attack was removed by the same means, as also a third attack some time later still. (*Exc. Med.*)

Gastric Catarrh of Children.—Dr. Müller (Posen) recommends the following simple prescription, especially in those cases where the motions are light in colour and of very foul smell: the dose given is for a child one year old:—

Tr. iodi.	..	℥v
Mucilag.	...	30 grams.
Syr. simpl.	...	20 "
Aq. dest.	...	50 "

Sig.: One teaspoonful to be taken three or four times a day.

A similar prescription, only with larger doses, proved very useful in the vomiting of pregnancy. (*Der Arz. Prak.*)

Cholera Infantum.—In very obstinate vomiting Dr. Rehfeld has seen most excellent results from

Emulsio. sem. papav.	...	100 grams.
Cocain hydrochlor.	...	0.1 gram.

Sig.: One teaspoonful to be given every hour. (*Therap. Monats.*)

THE CLINICAL JOURNAL.

WEDNESDAY, APRIL 8, 1896.

A CLINICAL LECTURE

ON FOUR CASES OF

PERFORATING GASTRIC ULCER.

Delivered at University College Hospital, March 12, 1896,

By A. E. J. BARKER, F.R.C.S. Eng.,

Professor of the Principles and Practice of Surgery at
University College, and Surgeon to University
College Hospital.

GENTLEMEN,—By a curious coincidence, I have had, within the last three months, four cases of gastric ulcer, which perforated and required surgical interference. I propose to make these cases the basis of some very brief observations in a clinical lecture. I am anxious to discuss the matter, as far as possible, from a surgical point of view, only touching on the physician's aspect of the case as briefly as may be. The most important part of all in the treatment of these ulcers is really the medical treatment before they come to the surgeon, because physicians ought to be able to prevent such things as perforating gastric ulcers by suitable treatment beforehand. But I want to emphasize, as far as I can, the matters which concern the surgeon when he is called upon to deal with these cases.

Until the last few years, perforations of the stomach by gastric ulcer were considered to be wholly out of the reach of the surgeon, and the earliest case of operation for the condition which I have been able to find in the literature of the subject, occurred about two years ago. That case was successful. Therefore, the surgical treatment is of comparatively recent growth, and we are still "feeling our way."

First, I would ask you to take carefully into consideration the class of patient in which these lesions are found. Gastric ulcer is essentially a disease of early life, and is usually found in persons of 15 to 25 years of age. Post-mortem records seem to show that the number of males and females attacked is about equal; while the clinical study of the subject leads us to believe that the majority

of the cases occur among females. I think this latter is the general impression. An analysis of 101 cases admitted into Guy's Hospital, quoted by Dr. de Havilland Hall, gives 59 males, 42 females, showing a slight male preponderance. Every one of the cases operated on, which I have been able to collect from published records, has been a female, and, in spite of the post-mortem statistics, I think we may safely conclude that gastric perforation, as we shall be called upon to treat it surgically, will occur more frequently in females than in males.

Referring more particularly to the four recent cases of my own, I ask you to note that the ages were respectively, 23, 21, 26, and 17—all practically within the range I have just mentioned. They were all servant maids, and it is a remarkable fact that almost all the cases on record of operation being done for this accident have been of the servant-maid class. Possibly, this may be accounted for by the fact that many of these girls are poorly fed before commencing their career as domestics, and that, with a wider range of dietary they commit indiscretions in the direction of overfeeding; and this, coupled with hurried, and possibly irregular meals, and inordinate tea-drinking leads to the condition.

The previous history of all these cases was also alike. They all gave an account of dyspepsia, pyrosis, pain after food, and discomfort; but, oddly enough, in none of these four cases were there the usually prominent symptoms of vomiting of food, or of blood. One or two of them had some regurgitation of food, but that was not a prominent symptom. They all had anæmia, pain in the epigastrium, dyspepsia, but no hæmorrhage. So far, they may have had very little to alarm themselves, their friends, or their medical man, and only a very skilled physician would have been on his guard, and on the look-out for danger, until the moment of perforation. Had there been marked vomiting with hæmatemesis, there would have been something to alarm their friends, and to have indicated the condition to any physician or surgeon, and thus led to different treatment from

that received by some of them. It must be remembered that a great many people do suffer from discomfort in the epigastrium, pain, pyrosis, eructation and anæmia, who have not got gastric ulcer. But once the symptom of hæmatemesis is superadded, the symptoms are very typical indeed. Therefore, I advise you to be always on the lookout for these earlier symptoms, and especially for any evidence of bleeding or contraction of stomach, with regurgitation of food or blood.

Once the rupture had taken place, the symptoms were so clear, as to leave practically no ground for mistake whatsoever. I will enumerate them. Sometimes the rupture comes on immediately after exertion, but in none of these cases does it appear to have done so. There was in all sudden pain in the epigastrium, gradually extending, in the course of a few hours, over the whole abdomen. Then there was in one or perhaps two of these cases, as is indeed usual, one or two attacks of vomiting. That is probably due to the faintness which supervenes as the result of shock from the bursting of the stomach wall; the fact that it does not occur more than once or twice, is probably because the expulsive efforts of vomiting drive the blood to the brain, and restore the mental equilibrium, and prevent subsequent faintness. Next, we found extreme tenderness in the epigastrium, on the left side, so that the patient could not bear the slightest touch on the part. Then there was rapid distension of the abdomen, which gradually became general, and obliterated the liver dulness—a most important feature in these cases. It was well marked on both sides, so that a clear note was obtained from the clavicle over the lungs on the right side, down to the abdominal area; on the left, the clear note was interrupted to some extent by the cardiac dulness, but after that there was no further interruption of resonance. Next, in all these cases there was a rapid rise of temperature as taken in the rectum, where one showed $104^{\circ}2$. Do not be misled by this, because though usual, a rapid rise is not invariable; and it is only in the earlier hours probably that the temperature rises high; subsequently, at all events, in a good many cases, it shows a tendency to fall. In the worst of these four cases with which I am more immediately concerned, the temperature had fallen to normal in the rectum within thirty-six hours. That indicates a condition which is alarming as a rule, and if

there is a subnormal temperature, the case is very grave.

Now, though the four cases were alike in all these respects, they were quite different in one or two most important features, from a surgical aspect. Three were alike, too, in having been operated on in the same theatre, and all by having been treated by the same surgeon, and in the same manner, and further—an important matter—were all under the care of the same house-surgeon. All these conditions influence the risks, because one man's methods may vary from those of another. But the most important points of contrast in these cases, consisted in the totally different periods of time which elapsed between the rupture and the application for surgical treatment. The first came into my hands at least 26 hours after perforation, the next $7\frac{1}{2}$ hours after, the next 28 to 30 hours, and the last, 36 hours after the accident. This is a matter which is of the utmost importance as bearing upon the operation. I may say at once that the only patient of the four who has not died is the one that was placed in my hands $7\frac{1}{2}$ hours after the accident; the one which lived for some fifteen days after, and nearly recovered, was in my hands for operation 26 hours after the rupture, while the worst case of all, and the one which succumbed soonest, presented the longest interval before operation. In support of this view, that the result is influenced by the time lost between rupture and operation, I may allude to a short list which I have compiled for my own use; this list shows that *only* the cases which were treated relatively early really recovered. That is the fact I want to impress upon you, and if by any means we can spread that notion more widely, by clinical lectures, by teaching, and by conversation, we shall do more good than any surgical arrangements we can possibly make. The operative procedure may be very perfect in itself, but if the patient is practically moribund, as two of these four were, at the time of operation, the chance of recovery is at vanishing point.

On opening the abdomen, we found exactly the same condition in all these cases. The perforation in all these was on the anterior surface, though the actual locality differed somewhat, and no great difficulty in closure was presented. They all had indurated margins, and the sizes of the perforations varied between that of a threepenny and

a sixpenny piece. In all there was great effusion. In the first, the whole upper part of the abdomen was full of fluid; in the second, the whole of the abdomen, from diaphragm to uterus, was filled with fluid. In the third, the same condition was found, but here there was a much larger quantity of lymph than in the preceding. And in the last case which came to me, thirty-six hours after the perforation, the amount of lymph was enormous, and was mixed with the contents of the stomach, such as cabbage, potatoes, etc.

Now, as to the operation. The method, with one slight difference, was alike in all. All were anæsthetised with chloroform; then, having cleansed the skin of the abdomen, and administered stimulating enemata by the rectum, I proceeded to open the abdomen. There is some difference of opinion as to the position and direction of the incision which should be made in these cases. In all the instances which I am dealing with, I began my incision by a cut in the middle line above the umbilicus, and in the last three, in addition to the median incision, I divided the rectus on the left side transversely. You may, of course, make a transverse cut straight away to begin with over the stomach, about two inches below the margin of the ribs. Do not go too close to the ribs, or you may find a difficulty in closing the opening, and their movements may interfere with its sound union; and if there be any suppuration in the wound, as is almost sure to be the case, the ribs may necrose, and give rise to a very serious condition. Perhaps the best incision is that which opens the abdomen in the middle line from the ensiform cartilage to the navel, supplemented by a transverse cut, which gives ample room to attack the stomach. This incision was made in another successful case which I have in my mind.

I will now briefly indicate the steps in the operation, laying particular stress upon the most essential points.

In the first place, there must be no delay in commencing the operation or completing it—the quicker it is done the better. The procedure is bound to be a lengthy one under any circumstances, therefore every fraction of time you can save will be so much help towards recovery. It must always be remembered that these patients are already in a condition of profound shock, and that must not be added to by delay. I need

hardly say that everything which may be required must be in perfect readiness before any incision is made. The patients, as in all my own abdominal operations, were wrapped in warm cotton wool from head to foot. After the vertical incision in the middle line, the cut across the rectus was made with one stroke of the knife. It may, of course, be made by transfixion with a bistoury. The hæmorrhage from the twigs of the artery can easily be checked. The moment you have opened the peritoneum, you find a great quantity of gas and fluid escapes, and after this has been released we get a return of the liver dulness. I found that very markedly in these cases. I percussed before the operation, and again after the escape of gas; in the former case there was resonance, in the latter dulness, showing the clear note to have been due to the presence of gas. The next point is not to begin by swabbing out the abdomen, but, first, to empty the stomach. This we did by squeezing the organ with the fingers, and the process enabled us to see the perforation. It is not always easy to empty the stomach through the opening, and some surgeons have thought it better to put a tube into it by the mouth, and flush it out with warm water. I have not done so in these cases, as it is possible that it might increase the delay, and thereby add to the shock. After clearing the stomach, I dusted iodoform round the opening, and not until then did I turn my attention to the general abdominal cavity generally. The first thing which I believe ought to be done in these cases, and which was done in three out of the four cases I am concerned with, is to make sure that the space between the liver and the diaphragm is perfectly clean. The neglect of that is the rock upon which many of these operations split; over and over again patients have had their abdomens cleared out, have recovered and lived for days, as in my first case, and yet have subsequently relapsed and died, as did my first case, on the fourteenth day, from the formation of an abscess between the liver and diaphragm, due to this part not having been clean. I do not think you can do better than slip your hand into the abdomen, between the liver and diaphragm, and wipe both surfaces of the space with a sponge repeatedly, thus bringing away any particles of food and lymph collected in it. Afterwards, work down by the spleen, which is very apt to have about its

mesentery a collection of fluid, then down by the kidneys and into the pelvis, wiping the intestines and behind the uterus. Then wipe the bladder, and work up the right side round the kidney to the liver, the right subphrenic space being cleansed like the left: then pay particular attention to the depression behind the gall bladder. In my first case we found, post-mortem, not only a subdiaphragmatic abscess, but a little collection of pus behind the gall bladder, in a kind of pocket. That may not be of very great importance, but it suggested to me a likely hiding-place for septic material. In the three subsequent cases, I cleansed that part with particular care. The abdomen being now cleaned as perfectly as possible, and the fluid having been previously squeezed out of the stomach, your next care is to stitch the perforation in that viscus. I think it is now pretty well settled that we ought not to excise the base of the ulcer; it has been done by many surgeons in the past, but those who have done it have recognized that it adds to the severity of the operation, increases the bleeding, causes delay, and does not facilitate the closure of the aperture of the stomach; moreover, it is not necessary. The stomach should be taken in the fingers and stitched, each stitch being passed into sound tissue, well wide of the wound, and arranged in such manner as to turn in the edges of the ulcer, bringing healthy tissue on both sides into contact. From eight to fifteen stitches suffice, and if care be taken to insert them well wide of the induration, one row of sutures will generally do. Remember, that if your stitches hold for six or eight hours, they will probably hold for ever; if they will not hold for that time, no double row will help you.

In the cases in question, sponges were left in the abdomen during the stitching of the perforation, and when they were taken out, the omentum was drawn up and laid over the line of suture, after the latter had been dusted with iodoform; and the intestines, which had been a good deal pulled about by the mopping, were laid in their places. Then followed a final mopping in both flanks, and the closure of the abdominal wound.

The question often arises whether we should completely close the abdominal wound, or put in a drainage-tube. In all these four cases of mine, I was obliged to drain. In the first, I closed the wound, feeling that if drainage were required it

would be indicated; it was required, and I put in a drainage-tube a day or two after operation. Some patients have recovered without any drainage, and, where the cases can be treated very early, possibly it can be dispensed with. Still, as a rule, it is wise to drain, and besides the tube, it is well to put in strips of iodoform gauze, laid in various directions; one would go from the middle opening in the abdominal wall up to and over the liver; another under the liver and over the stomach, another one side, and a fourth on the opposite side, the ends of all projecting from the median wound. These suck up the fluid by capillary attraction, and draw it into the dressing on the outside. These strips may remain in for a variable time; in the case which recovered, I took them and the tube out on the fifth day.

Now comes the question whether you should flush the abdomen. At first my own predilection when operating formerly upon somewhat similar cases of suppuration in the abdomen was decidedly favourable to general flushing. But I have lived through that stage, and I now feel that most of these cases are better without elaborate flushing. I believe a good many surgeons are coming to the same conclusion. Flushing will carry away a good deal of fluid, but it will not remove the solids, whereas wiping with a sponge will clear away both solids and liquids, as well as lymph. The abdomen will bear a considerable amount of manipulation in that way, when it cannot bear much flushing. In some cases two incisions have been made—one above the pubes—to provide an outlet while flushing, but I am bound to say I very much prefer to take away the lymph, food, and other material with sponges as quickly as I can. Remember, that if you put in a fluid, you have also to take it out again, which means sponging, in any event. If the fluid be left behind, it is septic in itself, and would form a focus from which inflammation would start. I should limit myself to flushing, with a jug of water, the area I had first exposed.

As to the material for stitching, most of us rely upon silk, both for the stomach and the abdominal wall; catgut, horsehair, and silver not being so reliable in my opinion.

The four cases which formed the basis of this lecture showed the following results:—

The first operated on about twenty-six hours

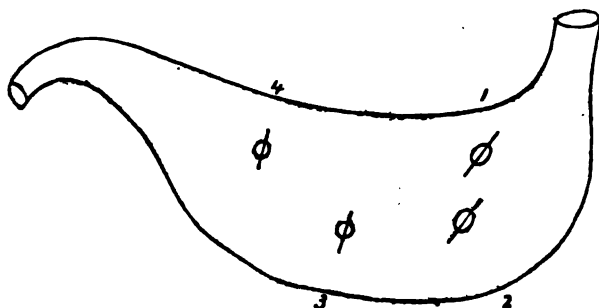
after perforation recovered well from the operation, the temperature went down, and she seemed on the way to recovery of good health during seven days. Then the temperature began to rise, and continued to do so until it reached 106° on the fifteenth day, when she died. I found a large abscess between the diaphragm and the liver on the left side (with commencing pleurisy on the upper surface of the diaphragm on left side), the remainder of the abdomen, except just near the wound, being perfectly normal. Before the onset of bad symptoms she was taking food well.

The next, which was operated on seven hours and a half after the accident, practically had not a bad symptom, and is now well and about, three months after the operation.

The third patient (operated on twenty-eight hours after perforation) was in a state of utter collapse, with a thready pulse at 140, and temperature 104° . Although she had benefitted by the opening of the abdomen, the escape of gas, and the removal of septic material, she had not enough strength to go through, and she died within the first twenty-four hours.

The patient whom I operated upon last also died, I regret to say. This is the case I saw thirty-six hours after the perforation occurred, and I think I may truly say that the operation did not hasten, if it did not favourably influence the final issue.

To sum up, there is a prospect of relieving persons who are unhappy enough to have a ruptured gastric ulcer, but if we are to cure them they must be brought to us within a reasonable time after the mishap. If this were done, I believe we should have a very large percentage of recoveries, instead of, as at present, a large proportion of deaths.



The marks on this diagram indicate approximately the position of the perforations (all on the anterior wall of the stomach) in these four cases as numbered. The stroke in each case indicates the line of suture for closing the perforation.

CLINICAL LECTURE

ON

CASES FROM THE THROAT DEPARTMENT.

Delivered in the Post-Graduate Course at the West-London Hospital on February 26th, 1896.

BY

JAMES B. BALL, M.D., M.R.C.P.

Cases of Lupus and Inherited Syphilis.

THE first case I shall ask you to look at is a girl suffering from lupus of the pharynx. She is 19 years of age. She first came to the Throat Department on December 14th, 1895. She gave a history of some soreness and discomfort in the throat for about ten months. The condition then was similar to what you see now, except that there has been some improvement. The soft palate, nearly as far forward as its junction with the hard palate, is studded with red granulations of various sizes, and pitted with numerous small ulcers, and presents cicatricial striæ in places. The uvula is elongated, thickened, and irregularly ulcerated, and presents many red nodules. Both faucial pillars and tonsils are irregularly pitted and nodular. On the left edge of the tongue, near the fore part, is a roundish patch of ulceration with an irregular surface. The larynx is normal, but with the laryngeal mirror you will see some large pale nodules at the base of the tongue on the right side. She has been taking cod-liver oil and arsenic since her first attendance, and there is decided improvement in the appearance of the throat—many of the ulcerated areas have cicatrized. As the case is improving, I have used no local treatment up to the present.

The next case is this girl, æt. 15. She has been attending since March, 1895. When she came there was a history of sore throat for eighteen months, and for about two months there had been hoarseness of voice and slight laryngeal dyspnoea. On examination, well-marked lupus of the soft palate and of the larynx was found to exist. There was a small patch of lupus on the left side of the neck, where a scar is now situated, and there was also some lupoid ulceration of the fore part of the

septum of the nose, and a small perforation of the cartilaginous septum. The condition of things is very different now. If you look at the soft palate, it appears quite normal, except for some fine cicatricial striæ in places. If you examine her with the laryngeal mirror you will see that the epiglottis is misshapen; apparently a portion of the tip has been destroyed. The fore edge towards the left side is thickened, and presents some large pale nodules. The ulceration in the nose has quite healed. The thickened and nodular condition of the edge of the epiglottis is the only sign of the disease remaining. The treatment adopted was the following. She was put on arsenic and cod-liver oil. The patch of ulceration on the neck and the ulcerated surface in the nose were scraped. No local treatment was used for the throat, which began to improve from the first. She was sent for six weeks last summer to the Sea Bathing Infirmary, Margate.

Before making any comments on these two cases, I should like you to look at this boy, who is suffering from inherited syphilitic ulceration of the throat. He is 9 years of age. He first came to the hospital last August, and the history of sore throat dated back only one month. The appearance of the throat at that time was very similar to the present condition. In fact, this is merely a relapse of the ulceration at the old site, due to his having remained under treatment for only a very short time. You see there is a deep cleft in the soft palate, the edges of the cleft presenting a dirty ulcerated surface. On the left side the ulceration has eaten deeply into the faucial pillars and tonsil. The whole of the ulcerated surface is dirty and sloughy, and there is much secretion in the throat. If you examine his eyes you will see well-marked opacities in both corneæ, characteristic of old keratitis. He is now taking iodide of potassium, and the condition will no doubt improve as rapidly as it did last August, when he first came under observation.

I have brought these cases before you to-day because they are in many respects good examples of the two diseases in question, lupus and inherited syphilis; and in chronic ulcerative disease of the throat, in young subjects, the diagnosis will mostly rest between these two diseases. In many cases of lupus of the throat, the presence of lupus on the skin will assist the diagnosis, but in the first

of these cases there never has been any appearance of lupus on the skin, and in the second, the patch on the neck did not appear till long after the commencement of the disease in the throat. Lupus is a disease essentially of youth. It generally commences about the age of puberty, but, of course, it may appear at any age from young childhood to adult life. The late ulcerative lesions of inherited syphilis often appear about puberty, rather before puberty, however, as a rule, and, indeed, one must be prepared for them any time from two or three years old onwards. The absence of pain is very noticeable in all three cases. Pain is not a prominent symptom in either disease. If we consider the course and rate of progress of the local destructive lesions, the two diseases are in striking contrast. The boy had only suffered from discomfort in the throat for one month when he was first seen, and yet there was present considerable destruction of the soft palate, which was slit right in two; and there was deep excavation of the tonsil. Contrast this rapid destruction with the state of things in our lupus cases. There is a definite history in the first case of about a year's sore throat, and yet the soft palate is not even perforated. There is no great loss of substance anywhere. In our second case the soft palate had been affected probably for a year or two before I saw her, and now that the disease has disappeared there is no deformity or destruction of parts, and except for the slight deformity of the epiglottis, the disease has left little trace behind. Of course, this is not always so, but it is the rule. This boy, fortunately, did not have any invasion of the nose. Had his syphilis attacked the nose, he would very probably have lost more or less of the bony structure. Lupus, on the other hand, never leads to destruction of bone.

The characteristic appearance of lupus of the pharynx is, as a rule, very different from that of syphilis, the difference being well exemplified in the cases I have shown you. In lupus you have the small reddish nodules, and the small, shallow, pit-like ulcers, the confluence of which produces serpiginous or worm-eaten areas; and the surface generally is clean and free from secretion. Syphilis, on the other hand, is characterized by one or more deep dirty ulcers, perforating or cleaving the soft palate, excavating the tonsil or posterior wall of the pharynx.

Collateral signs may be present, indicating the nature of the disease. The frequent co-existence of lupus on the skin has already been alluded to. The upper central incisor teeth may have the peg shape and notch characteristic of syphilis, or, as is the case with the boy we have just seen, there may be signs of old keratitis. In any doubtful case the administration of iodide of potassium will clear up the diagnosis.

Iodide of potassium will cure the ulceration of inherited syphilis. Perhaps the inherited disease does not always respond to it so quickly as the acquired form. If it should not respond rapidly, a little mercury may with advantage be added. Cod-liver oil is often a useful adjunct to the treatment. The most useful drugs in lupus are cod-liver oil and arsenic. Arsenic seems to be very useful in some cases. It should be given in full doses. In the second case I showed you, I curetted the nose, and the patch on the neck; but no local treatment was used for the throat, and it has got practically well.

In the first case there is already great improvement without local treatment. Some of these lupus cases exhibit a marked tendency to improve as soon as we attend to the general health. Many can get well in a comparatively short space of time with arsenic and cod-liver oil, especially if we can send them down to Margate for a time. Still we must be prepared for relapses and outbreaks in fresh sites. Some cases are very obstinate, and in these we must have recourse to active local treatment. The best local treatment is scraping with the curette, on the application of lactic acid, or a combination of both. The lactic acid is not to be merely painted on, but rubbed in vigorously with a small firm mop of cotton wool.

Case of Paroxysmal Sneezing.

This girl came to the hospital a fortnight ago, complaining that, for the last six or seven months, she has had almost constant "colds in the head." She is 22 years of age, is employed in a drapery establishment, and has always enjoyed good health. The colds come on every morning shortly after rising, and consist of sneezing attacks, running at the nose, and watering of the eyes. The attacks make her head ache. They pass off after half an hour or an hour. If you examine the nasal passages the only abnormality observable is a

ridge, projecting from the lower part of the septum on each side, running horizontally backwards from near the anterior end of the septum, for about an inch. On the left side this projection is jammed against the inferior turbinated body.

This girl is suffering from a very common affection. I have always several such cases attending the throat department, but I have selected her, as she exhibits the complaint in, what I may term, its most elementary form. It is a complaint which is to be met with in every degree of severity and in innumerable forms, and I may add that it has received a variety of different names.

It is a familiar fact that certain individuals are liable to be seized, at longer or shorter intervals, with coryzal symptoms, such as sneezing, watery discharge from the nose, injection of the conjunctiva, etc. The attacks develop suddenly, last a few minutes to some hours, and subside rapidly. The sneezing may be very violent and, in fact, it usually constitutes the prominent symptom, hence these cases are very commonly designated paroxysmal sneezing. On the other hand, the profuse serous discharge from the nose may be the main feature, and there may be very little sneezing, and such cases have been termed rhinorrhœa. This patient described her attacks as "colds," and this is what patients usually call them; but the rapid occurrence and subsidence of the attacks, their short duration, and the absence of constitutional symptoms, serve, among other points, to distinguish them from attacks of acute rhinitis. In addition to the symptoms just mentioned, you will find, in course of time, if you follow up these patients, that a certain number will develop, during the attack, or immediately after the attack, a little wheezing in the chest, and in time definite asthmatic attacks may supervene.

The essential element of the attack has been thought to be a vaso-motor disturbance in the nasal mucous membrane, and the term vaso-motor coryza has been suggested as a suitable name. Whether we term the complaint vaso-motor coryza, periodic coryza, nervous coryza, paroxysmal sneezing, or otherwise, we must recognize it as comprising a large group of cases, in which there is one common factor, viz., a peculiar hyperæsthetic condition of the nasal mucous mem-

brane. In a large number of cases the attacks occur without any assignable exciting cause, as in this patient. In many patients, as in this one, they are chiefly observed shortly after rising in the morning. In women they are sometimes noticed to be most marked about the menstrual period. On the other hand, in certain patients we can trace some exciting cause, and the possible exciting causes are almost innumerable. Dust of any kind is a frequent cause. The pollen of a variety of plants, the powder of ipecacuanha, colocynth, scammony, linseed, the exhalations from various animals, such as cats, dogs, rabbits, hares, etc., the skins of various animals, the odour of violets, and various scents, have all been known to act as exciting causes in particular individuals. Sudden changes of temperature will induce attacks in some persons.

Now as to treatment, and first, as to local treatment. If we examine the nose, we may find nothing abnormal, and in my experience these cases are the least hopeful for curing. Still even in these we sometimes get permanent benefit from the application of the galvanic cautery to the inferior turbinated body. In many cases, however, we find some pathological condition in the nose. In this patient there is, on each side of the septum, a prominent ridge running horizontally backwards for some distance, near the floor of the nose. On the left side this ridge is in firm contact with the inferior turbinated body. Deflections of the septum, or ridges and spines projecting from the septum, impinging on the inferior turbinated bodies, are the commonest local conditions met with in these cases. It seems that the prolonged contact of the septum, or of an outgrowth from the septum, with the inferior turbinated body, sets up in certain predisposed individuals a hyperæsthetic condition, which renders them liable to the symptoms this patient suffers from. Other pathological conditions may also bring about the same result, but then septal deformities are in my experience the commonest. What is certain is that we can cure, or greatly benefit, a great many of these patients by removing the source of local irritation. By correcting the septal deformity, or by removing the projecting spine or ridge, or by so treating the inferior turbinated body, that there shall no longer be contact between the parts, we often succeed in curing all the symptoms.

Can we do anything by general treatment? Certainly. The drugs I chiefly rely on are iodide of potassium, quinine, arsenic, and belladonna. This girl has been taking one grain of quinine and five grains of iodide of potassium for the last fortnight, and declares she is much better than she was. A useful prescription in this, and allied conditions, is a pill containing quin. sulph. gr. iss, arsenii iodid. gr. $\frac{1}{4}$, extract. belladonnæ gr. $\frac{1}{4}$. One pill to be taken three times a day. I have found this prescription very beneficial in hay-fever, which is a particular variety of the complaints we have been considering. As regards this patient I shall probably have to remove the projection from the left side of the septum. This will be most conveniently done with a fine narrow saw.

Case of Chronic Laryngitis.

This girl, whose age is 23, came to the Throat Department a few days ago. She is a healthy-looking girl, a domestic servant by occupation. She complains of hoarseness of voice, which has existed nearly a year. If you examine the pharynx—and you should always do this before looking at the larynx—you will notice that the posterior wall appears dry. You can see it is covered with a dry coating of mucus. If you were to take a little mop of wool and rub the surface, you would see that it is moist underneath, and that the dry appearance is due to a coating of dried mucus. Examination of the larynx shows decided congestion of the vocal cords, and on the inter-arytenoid fold there is a layer of inspissated mucus. If we now examine the naso-pharynx with the rhinoscopic mirror, we see some inspissated mucus adhering to the posterior wall—not so dry as that on the lower pharyngeal wall. The nasal passages, examined from the front, do not show any abnormality, except that on the fore part and under surface of the middle turbinated body there are some dry adherent flakes of mucus.

I have brought this case under your notice principally for the purpose of impressing upon you the necessity, in every case of chronic, or repeatedly recurring laryngitis, of looking beyond the larynx for the key to the chronicity of the complaints. I have no intention of entering into all the possible causes of chronic laryngitis, and I shall not dwell on the possibility of a chronic laryngitis having a syphilitic or tubercular origin,

or upon the effects of excessive use of the voice in producing the condition. But I want to impress upon you the necessity of always carefully inspecting the whole of the upper air-passages, the nose, the naso-pharynx and the pharynx, as well as the larynx, for here you will most assuredly find, in the majority of cases, a key to the chronicity of the laryngeal trouble, and in this way only can you direct your treatment to a satisfactory issue. Especially note a dry pharynx. You may find a variety of morbid conditions in the nose and pharynx, but any condition which is accompanied by a tendency for inspissated secretion to adhere and dry upon the pharyngeal wall, is a powerful predisponent to chronic laryngitis.

This dry condition of the pharynx—*pharyngitis sicca* it has been called—is often associated with nasal obstruction of some kind, but in this patient there is no nasal obstruction. You can tell this without examining the nose, by the fact that she breathes habitually with the lips closed. A dry pharynx is an almost constant accompaniment of atrophic rhinitis or simple ozoena, a disease characterised by a special tendency for the secretion to rapidly become inspissated and dry. In this patient there is merely a morbid condition of the glandular secretion, which renders it prone to dry and adhere to the surface of the mucous membrane, a slight dry rhinitis and pharyngitis. There is one feature in the appearance of the larynx which is always present in cases associated with pharyngitis sicca, and that is the collection of inspissated secretion upon the inter-arytenoid fold. I cannot say whether this is secreted *in situ*, or is caught up by the fold from the pharyngeal wall, but I think that in many instances the latter is the case.

In the treatment of these cases our attention must be directed simultaneously to the parts above the larynx, as well as to the larynx, otherwise we shall find that the laryngeal condition will not readily respond to treatment. In some cases we may have to deal with a deflected septum, hypertrophic rhinitis, ozoena, or other condition requiring special treatment, before a permanent cure can be insured. In most cases of chronic laryngitis associated with chronic rhinitis and pharyngitis, certainly in all cases associated with pharyngitis sicca, we shall greatly assist the treatment by the use of an alkaline detergent lotion, syringed or

sprayed into the nose so as to thoroughly moisten and cleanse the surface of inspissated secretion, and also used as a gargle or sprayed into the pharynx. I have ordered this patient the following: Sodii bicarb., boracis, sodii chlorid., āā gr. vii; sacch. alb. gr. xv. To be dissolved in half a tumbler of warm water, and used as a collunarium and gargle twice a day. For the larynx I have ordered the pharmacopœial fir-wood oil inhalation night and morning, and I shall apply a solution of chloride of zinc (30 grs. to the ounce) directly to the larynx each time she attends the hospital.

A CLINICAL LECTURE

ON TWO CASES OF

RECURRENT CALCULUS TREATED BY LITHOTRITY.

Delivered at Charing Cross Hospital by

JOHN H. MORGAN, M.A. Oxon., F.R.C.S.

GENTLEMEN,—On Friday last you witnessed the operation of lithotritry performed on two patients, whose cases though presenting many points of contrast, yet had one common feature, viz., that both had had stones removed from the bladder on previous occasions, though by different methods.

Let us first consider the details of the two cases separately, and we can then review the treatment and its results.

The first patient is a blacksmith, æt. 64, strong and healthy, with a good family history, and who has never suffered actively from gout or rheumatism. He believes that he used to suffer from gravel, but this did not cause him much trouble, until two years and a half ago, when he came under my care with all the symptoms of calculus, and on sounding, a stone was found on October 13th, 1893, lying on the right side of the middle line. This was seized by the lithotrite and rapidly crushed, and the fragments evacuated in the usual manner. Judging by the marks upon the handle, the stone as seized between the blades was about half inch in diameter. The fragments when dry,

weighed ninety-six grains, and were composed of urate salts only. He had no bad symptoms; but twelve days after the operation he went for a long walk, and on returning there was a slight tinge of blood in his urine, and accordingly he was again placed under the anæsthetic, and the bladder was again washed out. No fragments came away, and none could be felt or heard either by contact with the evacuating catheter, or with the sound, which was of course also used to explore the bladder. He remained perfectly well for upwards of two years, but four months ago his former symptoms began to return. He suffered, when admitted a week ago, from a good deal of pain, which shot down to the end of the penis, pain round the back and abdomen, and down the thighs. Distress was worse at the end of micturition, but was present before and during the act. It was worse during the day time, and especially on taking exercise. There was practically no pain when he was keeping quiet. He was passing water ten to fifteen times a day, and four or five times during the night, and exercise of any kind increased the frequency of the act. No blood has at any time been observed since the last operation, and on examination, the urine was found to be yellow and thick, alkaline, sp. gr. 1020, containing small amount of albumen, and some pus and mucus was found by the microscope. In all other respects he enjoys perfect health. *Crede experto*. Thus you will observe that the patient from his former experience, gave us all the symptoms which are so lucidly detailed by Sir Henry Thompson in the lectures, with which I trust you are all familiar, as pointing to the probable presence of stone in the bladder.

The symptoms thus accurately detailed, I deemed it unnecessary to sound the patient until he was upon the operating table. On January 23rd, he was put under the influence of æther, and as soon as the sound was passed, it hit upon a calculus which gave a clear sharp ring when the sound was brought in contact, which all those of you who were near could clearly hear. As on the occasion of the previous operation, the meatus required to be divided with a bistoury before the lithotrite could be passed, and as had been also found on the former occasion, the membranous portion of the urethra was slightly strictured. The largest and most powerful lithotrite would not easily pass, and therefore, one of medium size was used. With

the greatest gentleness and care this was passed into the bladder in the manner and with the precautions that I indicated to you at the time of the operation. The stone was grasped at the first attempt, and proved to be of about the size of a walnut. It was extremely hard, but yielded to the force of the lithotrite as soon as the screw was applied. Several large fragments were seized in a similar manner, and the lithotrite being withdrawn the evacuating catheter was passed, water was injected, and a large quantity of fragments withdrawn. This performance was repeated twice, until, on the third occasion, no more fragments could be withdrawn by the evacuator, nor could any be detected by the sound which was subsequently passed. The fragments showed that the stone consisted entirely of uric acid, and their collected weight amounted to 310 grs. The patient's condition next day was very satisfactory, though the urine first passed contained a large amount of blood, which, however diminished as the day wore on. On the second day the temperature rose to 101.2, the urine, still containing a little blood, was alkaline, and had an ammoniacal odour, and on the third day the patient being unable to pass his urine required a catheter, and this was followed by a rigor, during which the temperature rose to 104°. These unfavourable symptoms, however, quickly subsided, and on the fifth day succeeding the operation, the patient was passing his urine naturally, though it still contained a small quantity of pus. On February 4th, or twelve days after the lithotripsy, he was again placed under an anæsthetic, and the bladder again washed out. A few small fragments were brought away, after which, neither the evacuating apparatus nor the sound could detect the presence of any foreign substance in the bladder. Since this date the man has been perfectly well and comfortable, and has only remained in hospital in consequence of a curious and unforeseen accident. It seems that during the first operation his arm was allowed to hang over the side of the couch, and the pressure on the musculo-spiral produced a temporary paralysis of the muscles supplied by that nerve, which is only now slowly yielding to rubbing and galvanism. This, of course, prevents him from returning to his trade of a blacksmith, but he is so nearly well that he will be able to use his hand and arm as well as ever in a very few days.

The second patient for whom lithotripsy was performed on the same afternoon was a young Yorkshireman, who has recently left the Artillery. His present age is 25, his family history is good, and no members of his family are known to have suffered in a similar manner. About 1890, when out riding he experienced a sharp pain all along the penis, and in the lower part of his abdomen, the pain being so great as almost to cause him to faint. The pain continued for about a week, during which he kept quiet, but when he began to get about again it became more intense, and although able to pass a good stream of urine, this would suddenly stop, and the pain became excruciating, and lasted for several hours. In January, 1895, he came under the care of Mr. Johnson Smith at the Seaman's Hospital, who has kindly favoured me with some particulars of the operation which was performed on January 17th, 1895. As he was then in poor condition, and suffering much from cystitis, Mr. Johnson Smith did what he considered the safest operation, viz., suprapubic lithotomy. The stone, which I have by the kindness of that surgeon seen, consisted of phosphatic material, and weighed 338 grs. Convalescence lasted three months, and by the extent and breadth of the cicatrix was probably attended with some suppuration. It is about three inches long, and about one inch at its broadest part. In March last, he joined his regiment again, and in September last, when taking part in a route march he experienced a good deal of pain in the penis, abdomen, back and thighs. On passing urine, he noticed that it was blood-coloured and thick, and towards the end of the flow a thick whitish material came away, the stream suddenly stopping and giving intense agony. The pain continued till October 15th, when he arrived at Woolwich, and on October 22nd he went into hospital, where he remained until Tuesday, January 14th, when he came to this hospital. During his residence in hospital at Woolwich, the desire to micturate was very frequent. The bladder was washed out every other day, he was given a good deal of medicine, morphia was injected, and hot fomentations were applied.

I sounded the patient without an anæsthetic, and at once found a fair-sized stone. The patient being under the influence of æther on January 24th, I introduced a medium-sized lithotrite and seized the stone, which was about the size of a large

filbert. The lithotrite and evacuating catheter were both introduced twice, and after the second occasion no trace of fragments could be found. The components of the calculus consisted entirely of phosphatic material, and weighed 130 grs. There were no drawbacks to his convalescence, but following my usual practice I washed out the bladder again on the twelfth day after the operation, and as no fragments were found he was in a few days discharged from hospital.

The opportunity of performing lithotripsy upon two patients on the same day does not frequently occur now that so many of these cases are treated in one way or another at local hospitals. You will remember the remarks which I made at the operation regarding the difference of method in the introduction of a lithotrite to that followed in using a catheter, and this difference was exemplified more particularly in the first of the two cases, where, by reason of a certain amount of constriction of the urethra greater care was required to be exercised, and greater patience. This point cannot be too strongly emphasised, and it is always painfully present to my mind from the fact that I have on two occasions been present when a lithotrite has been pushed through the walls of the urethra into the cellular tissue of the pelvis, with naturally a fatal consequence.

But leaving other points aside for the present, the first subject for our consideration in regard to these two cases is the fact that in both the stone was recurrent. Different operations had in both cases been practised for the removal of the previous stones, but the patients had in each instance suffered from a return of the malady, and in each case the nature of the calculus in the second instance was identical with that which had been removed on the former. Now, with reference to the first patient, for whom I had performed lithotripsy two years and a half ago, it might be urged that some fragment of the *débris* had been left behind, and had formed the nucleus of the second calculus. I do not deny that this may have been the case, but every precaution was taken to ascertain that such a fragment was not present, even to the extent of a second washing out of the bladder, and the use of the sound a fortnight after the operation. This is one of the principal objections to the operation of lithotripsy, and it is no doubt true, as Mr. Cadge has pointed out, that

repeated washings will still fail to discover a last fragment in some bladders.

This is more especially the case in patients with enlarged prostate, which certainly was not the case with the man in question. Moreover, the man passed two years in perfect comfort before noticing any return of his symptoms. Had there been a fragment left behind, this could hardly have happened. Moreover, if the second calculus had been due to the irritation of the bladder by a fragment of the former stone, besides suffering from the symptoms of cystitis, we should almost certainly have expected to find that the calculus removed the other day consisted in great part of phosphatic material. On the contrary, the fragments removed consisted entirely of lithic acid, nor was there anything to indicate that these had collected around a relic of the former calculus. There are three main sources for the formation of stone. First, the descent of a stone from the kidney, generally of uric acid, around which fresh deposits of the salt are accumulated in the bladder. Secondly, from immediate primary precipitation in the bladder itself, consequent on stagnation in the bladder, or otherwise; or, thirdly, it may be produced by the presence of a foreign body in the bladder, whether introduced from without, or existing within in the shape of coagula of blood, fibrin, etc., which leads to abundant precipitation. (*Donkin.*) In both of the two latter circumstances phosphatic materials enter largely into the formation of the calculus, and as stated, none were to be seen in the fragments which were extracted. It is therefore reasonable to suppose that this calculus which had probably formed within the last six months, had originated like the previous one in the kidney, and passing down into the bladder had there, by accumulation of fresh material, acquired the size of a walnut, and a weight of 310 grains.

Turning to the cause of the occurrence of a stone for the second time in the bladder of the younger patient, we learn from the letter which Mr. Johnson Smith kindly sent to me, that the man was suffering, at the time that he came under his care, from cystitis of some long standing, and was in poor condition, and under those circumstances, no doubt, any surgeon would have selected the operation of suprapubic lithotomy, which was so successfully performed by Mr. Smith. The patient returned to his duties, and probably

neglected to observe those precautions which were, no doubt, given to him, and in the course of six months began to suffer again from the symptoms of calculus, of which he was relieved by lithotripsy on January 24 of this year, just twelve months after the first operation. Now, as a result of the suprapubic operation, it may be asserted that in this case there could have been no fragment left behind to form a nucleus for a fresh deposit, and the occurrence of a second calculus in so short an interval must be traced to a different cause to that which probably gave rise to it in the elder patient. Here, the second stone, like the first, consisted entirely of phosphatic material, and was, therefore, due to an unhealthy condition of the urine, and an altered state of the mucous membrane of the bladder, remaining as a sequel to the former cystitis. It is more than likely, also, that besides the chronic change in the mucous membrane, the bladder itself may be very different from the normal as a consequence of the operation which has been performed. Even where primary union occurs, and where the incision in the bladder wall heals within a few days, as it often does in young subjects, there must remain a cicatrix which interferes more or less with the due contraction of the bladder, and prevent it from fully discharging its contents, and therefore tends to cause the presence of a certain quantity of residual urine, which is the most frequent cause of phosphatic deposits. But in this case, besides the cicatrix in the viscus, there is almost certainly adhesion of the bladder to the anterior wall of the abdomen as a result of the long process in healing, and this constitutes a further obstacle to the full and efficient discharge by the bladder of its contents. I have warned this patient that he must watch very carefully for any sign of returning trouble, and I shall not be surprised to hear of his reporting himself here or at some other hospital within the course of a few months with all the symptoms of a fresh stone. When he left the other day his urine was clear and acid, and if he could be induced to wash out the bladder regularly for some time to come it is probable that he would avoid further trouble. I think that this constitutes a serious objection to the suprapubic operation which has hitherto been overlooked. Since the reintroduction of the operation some twelve years ago it has been freely practised under the idea that, with modern ap-

pliances and antiseptic precautions, it was easy and free from risk, and it is recommended to be used by those who only have to deal with stone occasionally. From my own experience I strongly dissent from this view. It remains for someone to collect the statistics and results of this operation, and I believe that it would be found that, even in simple cases, the risks were considerable and the deaths many more than is supposed. The operation, as modified, is a most valuable one in certain cases where lithotripsy is not available, but for such, in my opinion, it should be strictly reserved.

You will naturally ask why, with the various operations that were available in both these cases, I chose the operation of lithotripsy? In the first case, a stone had recurred after lithotripsy, and it might be thought that this implied a failure of the operation to get rid of the stone. But I have given you what appear, to my mind, as sufficient and ample reasons for believing that the whole of the fragments were extracted, and that the second calculus was formed, like the first, by accumulations in the bladder around a stone that had passed down from the kidney. The patient has a strongly-marked lithic acid diathesis, and it is quite possible that, in the course of time, he may again suffer from the same malady. On the other hand, the second patient had had all the advantage which can be claimed for the suprapubic operation, and not only has the calculus rapidly recurred, but in his case also, from the condition of his bladder, I shall be much surprised if sooner or later he does not again suffer in a similar manner. The causes in both cases are different; and were they private patients, and constantly under observation and able to carry out proper precautions, it is possible that the eventualities predicted might be partially or entirely avoided. But I selected the operation of lithotripsy in preference to the lateral or the suprapubic method of lithotomy, because, as I believe, I was able entirely to rid the bladder of all foreign material, and this with little or no suffering to the patients, with no loss of blood, and with no injury to the walls of the bladder. Both patients were up and about in the course of a very few days, and were passing normal urine without pain or difficulty.

It has been my privilege and great fortune to see a great deal of the work of Sir Henry

Thompson, and anyone who has seen the extraordinary skill and rapid manipulation of the lithotrite in his hands may well be excused a preference for this form of operation, where circumstances favour the adoption of the crushing operation with the removal of all fragments at one sitting, according to the modern practice, which goes by the name of litholapaxy. You have seen the favourable results of this operation in these two patients, and there is at the present moment under my care a patient who was admitted last year with a tight and long-standing stricture and a large phosphatic concretion. The stricture was treated by rapid dilatation, and the stone crushed, and all fragments were washed out by means of the evacuator, which I have used for many years, and which I designed and described in the *Lancet* some fifteen years ago. With its mechanism and with its mode of application you are all familiar, as you have seen it used on many occasions, and like all other similar apparatus it serves the double purpose of evacuating the bladder and of distinguishing the presence of fragments which are too large to pass through the eye of the catheter. This third patient has returned with some symptoms of irritation of the bladder, but on passing a sound no calculus could be detected, and what is equally satisfactory, the urethra is so far patent that it offers no resistance to the passage of a sound, and the stricture is therefore as far as possible cured. The symptoms for which he has been readmitted are due to a slight cystitis, which will no doubt soon succumb to rest and treatment.

CLINICAL

DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London
Clinical Society, North-West London Hospital,
March 18th, 1896.

DR. CAGNEY in the Chair.

A Case of Multiple Neuritis.

Dr. CHARLES CLAYTON showed a patient, æt. 29 years, very active and athletic, of temperate habits.

Never had syphilis. Scarlatina as a child, not followed by dropsy; ten years ago an attack of pain and swelling in right great toe (? gout), and a similar attack twelve months ago. His father suffered from gout.

Came last October, in first week of typhoid fever. Characteristic, but rather mild course—in bed five weeks. After a few days in bed, sharp severe pains commenced in neck, shoulders, arms, and forearms on both sides, but more severe on the right. Pains lasted in an acute form for about a week, when they lessened; but even now they are occasionally felt, especially in neck. Soon after, spots of tenderness noticed in various places, and some patches of numbness in forearms, over one elbow, and in neck. When he got up, he found his arms very weak, and he was unable to lift them above level of shoulders. After a fortnight at the seaside, gradual gain of power in arms. Returned to town, and resumed habit of dumb-bell and other exercises, but suddenly one day noticed something wrong with right shoulder-blade.

His condition then (late in December) was as follows:—

Unable to raise right arm above level of shoulder; attempt to do so at once revealed deformity characteristic of paralysis of serratus magnus. Wasting, to greater or lesser extent, of following muscles: supra- and infra-spinatus, anterior portions of trapezii, triceps, and latissimus dorsi on both sides—deltoids very slightly, if at all, wasted. All showed diminished or abolished faradic and increased voltaic excitability, with readier response to anodal opening and closing contraction than to cathodal closure. A patch of anæsthesia over point of right elbow and along posterior surface of the same arm, another on the right side of the neck, and others on the front of each forearm. Touch was not perceived, but sensation of pain and temperature persisted at all these spots. There was tenderness over some of the nerve-trunks, especially over the suprascapular notches, and at a spot on the surface of the right serratus magnus, which appeared to be exactly over the course of the posterior thoracic nerve. Some of the muscles were also tender, but those of the forearms were unaffected. The grip, measured by the dynamometer, was over normal. A slight transverse groove was present on nearly all the finger-nails. The lower limbs were norma

as were also the cranial nerves, the viscera, and the urine. There was a blue line on the gums.

Dr. CAMPBELL suggested that the lesions might be in the anterior cornua of the cord—an anterior poliomyelitis, in fact.

Dr. CAGNEY discussed the distribution of the paralysis, and pointed out that the muscles affected were not those usually attacked in multiple neuritis. He commented especially on the immunity of the extensors in the forearm and of the deltoid, saying that both were eminently apt to suffer. Again, he was uncertain how far the somewhat slight sensory disturbance was to be associated with the motor trouble. Further, the paralysis of the serratus magnus—a rare lesion—on one side, and its subsequent recovery, were somewhat anomalous. He hesitated at first sight to accept the case as one of multiple neuritis. In favour of such a diagnosis, there was undoubtedly an adequate cause in the preceding typhoid fever, in the symmetrical arrangement of the paralysis, in the presence of some, even though slight, sensory trouble in the muscular tenderness, and a questionable trophic change. Against it was the difficulty of conceding that the neuritis was peripheral, as multiple neuritis generally is. The total palsy of so large a muscle as the serratus magnus could hardly be due, under the circumstances of the case, to such a course; but this, perhaps, was due to another injury, and was a coincidence. The distribution of the paralysis was also the reverse of characteristic. The alternative of an anterior poliomyelitis suggested itself, but this was to ignore the sensory disturbance. Symmetry was not so great an objection, but counted for something. Wasting of the scapular muscles was a notable incident in one form of the disease in adults, but in this form the upper part of the trapezii wasted here was the last to linger—had, in fact, been called by Duchenne the ultimune moriens. Finally, recovery, progressing here, was rare in these cases. In conclusion, he would notice that the arrangement of muscles selected by the paralysis was a physiological arrangement. It was not quite that of Erb's paralysis, but he believed that there, as in this case, this physiological arrangement gave a clue to the condition. The lesion may be spinal without being a myelitis; it may also be focal and multiple, or diffused, though not extensive.

M. Brissaud had speculated about such lesions, a true neuritis affecting the nerve fibres at their origin in the cord. The fact of symmetry there would not be more remarkable than in peripheral neuritis, and a physiologically-selected paralysis suggested it. For the benefit of the wasted muscles, he strongly advocated the intra-muscular injection of strychnine, in addition to galvanism and other measures.

DR. CLAYTON in reply expressed agreement with Dr. Cagney respecting the paresis of the serratus magnus—it was probably due to the too early resumption of exercises. The posterior thoracic nerve was especially liable to be injured by violent exertion, especially by overhead movements of the arm; the nerve ran a very long course, and was perhaps compressed by the scalenus medius muscle, which it pierced. Most cases occurred on the right side, the case was therefore according to rule.

The question as to whether the lesion was in the anterior cornua of the spinal cord, or in the nerves themselves, was, he considered, not very difficult to decide.

The rather gradual onset with acute shooting pains, the patches of anæsthesia, the tenderness of muscle, and over the nerve trunks, and the symmetrical distribution of the affection all pointed to the nerves as the seat of lesion. In anterior poliomyelitis the onset was much more acute, the paralysis was widespread at first, soon becoming more limited; the distribution was generally asymmetrical, and there was an entire absence of sensory symptoms.

The presence of a blue line on the gums, very like that of lead, suggested at first the possibility of lead-poisoning, but the freedom of the extensors of the wrist, the absence of any evidence of lead contamination, of anæmia, and of colic, rendered this supposition improbable. The patient had used charcoal as a dentifrice for fifteen years, and that, he was told by a very able dentist, is a frequent cause of a blue line closely simulating that of lead.

Loose Body in Knee-joint.

MR. DURHAM showed a piece of articular cartilage from a private in the 4th Hussars, æt. 26, who slipped down December 27th, 1895, striking left knee. He then had acute synovitis, which was treated by a plaster case, and subsequently

by blisters. Was admitted on February 15th, when an irregular hard, slightly moveable body was felt on outer and anterior aspect of left knee-joint, between tibia and femur, lying flat against skin. Was operated on, on February 17th, 1896. The joint was now almost normal again. The fragment formed an isosceles triangle, the sides being one inch, and the base half inch. There was a thin layer of bone beneath the cartilage, which appeared to have come from the patella.

MR. GORDON BRODIE confirmed Mr. Durham's view.

DR. COODE ADAMS pointed out that this case was really an unusual form of fracture of the patella, and differed from the ordinary cases of loose bodies in joints.

Nervous Impairment after Injury to Elbow.

MR. FREDERICK DURHAM showed a boy, æt. 13, who was admitted on January 21st, 1896, suffering from a severe lacerated wound of back of right elbow, caused by a circular saw.

The triceps muscle was completely severed, and piece of external condyle shaved off, and the whole joint completely opened up, and the synovial fluid had all escaped. There were two or three spurting vessels on removing the ligature above the joint.

MR. DURHAM operated, irrigating the wound with sterilised water only. He stitched the synovial membrane, then the triceps, and afterwards completely closed the skin without drainage. On January 22nd, the temperature rose to 101.6°, dressings were removed, and the joint found to be exquisitely tender and greatly distended. Two stitches were removed from the skin, but no fluid escaped. The joint was then aspirated, and two or three drachms of clear serum removed, and the joint was covered by a warm carbolic dressing. After this the wound healed rapidly. There was no suppuration. The patient has now perfect use of the arm, and there is only the slightest impairment of function in the ulnar nerve.

DR. SAVILL thought, in this case, there was definite evidence of partial injury of the ulnar nerve in the weakness of the ulnar muscles, flattening of the hypothenar, and the flushed condition of the inner half of palm, which was bounded by a somewhat abrupt margin. But he advised postponement of an operative procedure, because, he had seen and recorded similar cases, which, by rest, massage,

and galvanism had recovered in course of time. He also referred to Hilton's *Rest and Pain*, where other cases were described. If, however, a definite swelling could be felt in the course of the nerve, it might be well to cut down on this.

Dr. CAGNEY thought that there was practically no evidence of ulnar paralysis. A good gauge was the condition of the interossei, and they and the other small muscles of the hand were clearly intact. If, with Dr. Savill, he were to take some wasting of the flexor carpi ulnaris as showing an injury to the nerve, this could only be of the character Mr. Durham supposed, namely, that a small part of the nerve was cut. If that were so, while the rest was intact, the injured part could not be put in a better position for repair, which, indeed, should be well in progress by now. Practically, the only change he could discover at present was sensory anaesthesia, and this was recent and progressive. The fact pointed to a slight, a recent, and a progressive lesion. Such might be found in the pressure caused by the contraction of the scar of operation. This might need to be dealt with later. Operation on any other ground did not seem to him to be indicated.

Deformity of Forearm, following Fracture of Forearm.

Mr. JACKSON CLARKE showed a boy, *æt.* 12, who ten months ago sustained a fracture of the humerus one inch and a half above the elbow. When first brought to him, several days after the injury, on removing the splints there was seen much swelling above the elbow-joint, and sensory and muscular paresis of the forearm. Under electrical treatment and massage, conducted by Mr. Hosford, the case did well, though the supervention of hyperæsthesia combined with the neurotic temperament of the boy rendered this difficult. But, although at the end of six months no deformity was present, there was evidence of paralysis in the superficial flexor muscles and pronator muscles, and at the same time a nodal swelling could be felt on the median nerve. Mr. Clarke advised the boy's admission to hospital, but the boy's father objected. At the present time there was considerable deformity, the fingers being hyper-extended at the metacarpo-phalangeal, and flexed at the inter-phalangeal joints, all movements of the wrist and hand were limited. Mr. Clarke proposed the excision of the swelling on the median nerve. The

fracture had healed without any perceptible deformity.

Dr. SAVILL viewed this as a very serious case, calling for prompt surgical interference.

The spasmodic contracture and atrophy of the flexor muscles of the forearm, thenar and hypothenar pointed to a severe irritative lesion of both ulnar and median nerves, albeit sensation was not absolutely abolished. It seemed to him that these nerve trunks had been involved in the callus or inflammatory products at the time of the fracture, without the necessity for any hypothesis of a reflex irritation through the spinal cord, and that the forearm would not only become useless, but require removal, unless some operative relief were afforded.

It might be interesting to mention that he (Dr. Savill) had some years ago removed as much as *an inch and a half* of the course of the median nerve in the upper arm with impunity. The ends were brought to within five-eighths of an inch of each other by means of chromic gut ligatures. For a time the patient had considerable pain on extension of the elbow, *i.e.*, stretching the nerve for a long time, but ultimately he made a good recovery. He was now at work as an omnibus conductor. The only difference in the functions of the arms and hands was that he could not always detect the difference between a sixpenny and a shilling piece. This was, the patient declared, not from want of power to feel, but because his fingers seemed stupid, that is to say, there was a slight loss of muscular sense in the approximation of the first and second fingers of the affected side.

Dr. CAGNEY was not entirely satisfied that the case was one of paralysis of the muscles supplied by the median nerve. He thought, rather, that the position of the hand, and other considerations, showed paralysis of the extensors, and resulting contracture of the flexors. The matter was complicated by the account of a former anaesthesia of all surfaces of the hand and forearm. To produce this, the ulnar and musculo-spiral, as well as the median, needed to be involved. Confining himself to the state of things then present, he recalled a similar case pronounced by several eminent surgeons to be, infantile paralysis. It had its beginning in an injury precisely similar to this, and in a boy of like age.

(To be continued).

THE CLINICAL JOURNAL.

WEDNESDAY, APRIL 15, 1896.

A LECTURE ON THE DISEASES KNOWN AS ENDO- METRITIS.

Delivered at the London Hospital.

BY

G. ERNEST HERMAN, M.B. Lond., F.R.C.P.,
Senior Obstetric Physician to the Hospital, etc., etc.

THE late Dr. Matthews Duncan in the beginning of his lecture on endometritis says: "Who can tell what any one means by endometritis? Often its use is the parent or the child of ignorance and confusion: often it is a cloak of ignorance and confusion."

These statements are true for the following reasons. The endometrium cannot be seen or felt without trouble both to patient and doctor. Hence its condition is generally guessed at. It undergoes a monthly cycle of change; and we imperfectly know what these changes are. Without perfect knowledge we cannot say whether slight changes in the endometrium are pathological or physiological. Our knowledge being thus imperfect, and of there being changes in the endometrium at all in most cases conjecture, there is, as Duncan said, ignorance. Many people think endometritis a common disease, because they call any case by this name in which there is whites and increased menstrual flow. Therefore there is confusion.

If we are ignorant, the first condition of learning is to be aware of our ignorance. To label cases we know nothing about with a name tends to perpetuate ignorance.

There are three conditions known as "endometritis," which are different from one another. These conditions are (1) adenomatous growth, (2) catarrh, (3) what is called "atrophic endometritis." They cannot be distinguished from one another until the cervix has been dilated. I

describe them under the name of endometritis because they are so called in books. I shall give reasons for questioning the appropriateness of this name for some of them.

The first disease I have to describe is adenomatous growth.

Simple overgrowth of the endometrium is often spoken of as "*endometritis*." But the reasons for thinking there is inflammation are not conclusive. Inflammation has been defined as the response of living tissue to injury. One of its features is that when the cause of inflammation has been removed, an inflamed part is restored to its normal condition, as far as the damage done will permit. When mucous membranes are inflamed they secrete pus; and when microscopically examined, the changes which accompany inflammation are seen. Genuine endometritis,—that is a morbid change which presents these features,—which gets well when its cause is removed, and secretes pus, is seen sometimes, but does not always cause great hæmorrhage. What is called *fungous*, or *fungoid*, or *villous endometritis*, presents neither of these characters. It is an adenomatous or myxomatous growth, and often both kinds of growth exist together. I therefore speak of it as *adenoma* of the body of the uterus. We cannot distinguish, until after removal, adenomatous growths from myxomatous, and if we could it would make no practical difference.

There are two forms of adenoma of the body of the uterus: the *polypoid*, and the diffuse, or the *hyperplastic*.

The polypoid form is sometimes spoken of as "endometritis polyposa," "metritis interna villosa," "mucous polypus of the body of the uterus," "fungoid degeneration of the endometrium" or "uterine fungosities." It may be proper to apply also the term endometritis to these cases because the presence of these growths does excite a little inflammation of the endometrium; but the main morbid change is not inflammation, but new growth. In this form there are outgrowths from the mucous membrane of the body of the uterus. They are from the size of a pea up to an

inch in length. There may be only one, or the uterine cavity may be filled with them.* When examined microscopically they are found to consist of gland tissue, quite healthy excepting as to quantity, vessels, and connective tissue between, this tissue being looser in some places than others, and in some quite myxomatous. Some consist mainly of gland tissue; in others the connective tissue preponderates. They thus resemble, as to their structure, the common mucous polypus of the cervix; but while such growths are common in the cervix, they are rare in the body of the uterus.

When these outgrowths are large, they are often malignant: grow fast, and return after removal. Small outgrowths are seldom malignant. If the growth be larger than a hazel nut, malignancy should be suspected; the growth should be examined microscopically, and if the microscope shows only adenomatous structure the patient should nevertheless be watched, and the interior of the uterus again examined if there be any return of symptoms. There is no way of distinguishing a malignant from a benign adenoma, except by watching the case and observing the rapidity of growth and its return after removal.

In the *diffuse* or hyperplastic form there are no localised outgrowths, but the whole lining membrane of the uterus is thickened, softened, and easily detached. The finger can detach the mucous membrane in large pieces. When examined the structure is like that of the polypoid form; overgrowth of healthy gland tissue and vascular connective tissue between.

Both the polypoid and the diffuse forms are rare. We know nothing of the etiology of either. Among the causes that have been assigned are subinvolution, retroversion, and retroflexion of the uterus, and lacerations of the cervix. But these conditions are very common, and uterine adenoma is rare; and we know nothing as to why adenoma should occur in one case of subinvolution or retroversion of the uterus, and not in others. But to think that when the uterus is not quite in a normal condition new growths should be apt to occur, is in accordance with the little general knowledge we have about the circumstances of origin of new growths. The associa-

tion with laceration of the cervix is an American theory unsupported by the slightest evidence. Imperfect sexual intercourse has also been put forward as a cause, but without proof. The disease has been ascribed to "ovarian influence"; but as we have no means of detecting or controlling this "ovarian influence," the theory, if true, does not help us. With fibroids a similar condition of the mucous membrane may coexist; and this is a reason why we should dilate the cervix, and explore the uterus before urging that the patient be spayed.

In both forms there is some expansion of the uterine cavity, and therefore slight enlargement of the whole uterus: but the cavity seldom measures more than three inches and a half long. The uterus is quite moveable. There is, as a rule, no disease of the uterine appendages or of the cervix, although morbid changes of this kind may be accidentally associated with it.

The symptoms are hæmorrhage and discharge. I do not know that the hæmorrhage has caused death; but it often produces serious anæmia. It bears no relation to the number or size of the fungosities. The hæmorrhage may keep nearly to the monthly period, being then prolonged and excessive; it may come on irregularly, or it may be almost continuous. No inference can be drawn from its time of occurrence. Between the hæmorrhage there is generally discharge, not usually purulent, but watery and pink. There may be a little pain; but these patients come for treatment, not for pain, but for hæmorrhage. The disease generally occurs in sterile women. It is said to produce sterility, but I know of no evidence of this. Cessation of child-bearing usually precedes the disease. Beside these local troubles, there will be such remote symptoms as anæmia produces.

The *diagnosis* cannot be made without exploring the uterus. When the finger is in the uterine cavity we shall feel in the hyperplastic form the thick, soft mucous membrane; perhaps coming away in large pieces merely from the movement of the finger. This might be taken for an early pregnancy. If this be the nature of the case, in examining the membranes removed, we shall see the amnion, identified by its thinness and transparency. Nothing like this is present with an adenomatous growth. A uterus lined with the

* See Goodell, "Lessons in Gynecology."

decidua of an extra-uterine pregnancy presents a close resemblance to one containing diffuse adenoma; for in both there is overgrowth of the endometrium, and the thickened endometrium is easily detached. Therefore in any such case examine carefully not only the uterus, but the ovaries and tubes.

In the *polypoid* form we shall feel the polypoid growths in the uterine cavity. They will be distinguished from fibroids by their greater softness, their being more easily detached; and after detachment by cutting them open, when it will be seen that they are not made of white fibrous tissue.

It is not possible to distinguish before removal and examination an adenomatous polypoid growth, independent of pregnancy, from a small placental or fibrinous polypus, or a malignant disease following pregnancy, such as German writers have described under the name of "deciduoma." If there be a history like that of abortion, the nature of the growth may perhaps be suspected, but the history may be too indefinite to help us. It is not of importance to make the diagnosis between the two former conditions, because the treatment is the same; and if the lump be the remnant of a miscarriage the prognosis is more favourable than if of any other nature.

Deciduoma is distinguished by its microscopical characters, which are those of malignant disease; masses of epithelial cells of various shapes and with large nuclei invading the surrounding tissues. It is rapid in its course. The only treatment is removal of the uterus; which at present in this disease has been very unsuccessful.

The diagnosis between a *simple* and a *malignant* adenoma cannot in all cases be made with certainty. The more numerous and large the polypoid masses, and the more rapid the growth, the more likely is the disease to be malignant; while a single small polyp is probably innocent. But cases have been recorded in which large and multiple growths proved innocent; and a malignant growth is at first small. Therefore all that can be said is that, the larger the growth, the more careful should the prognosis be.

Treatment.—The treatment is the removal of the diseased endometrium. This is accomplished by scraping away as much as possible with the curette, and destroying the rest with caustic. The best *curette* is one which has a broad blunt end,

and is rigid. A curette like Marion Sims's, having a sharp end, may cut into the uterine tissue and cause troublesome hæmorrhage. A flexible curette bends, so that the operator cannot tell what he is doing with it.

Douche the vagina with 1-2000 sublimate solution. Let the volsella and the curette lie in the sublimate solution while this is being done. Seize the cervix with the volsella, and draw it down. Then with the curette scrape the anterior and posterior uterine walls, so as to detach as thoroughly as possible the lining membrane. This is often enough, but not always. It is probable, but not certain, that greater security against recurrence is obtained by destroying the superficial part of the lining membrane with caustic. The best caustic is nitric acid. Pack wool soaked in solution of carbonate of soda underneath and on each side of the cervix, which is held by the volsella on the anterior lip. Then take a piece of cotton wool in the grasp of a pair of forceps with thin blades, and roll it round the blades, so that it forms a mop about the thickness of the little finger, and that the wool projects about $\frac{1}{2}$ of an inch beyond the tip of the metal. Dip it in the acid, and pass it through the cervical canal up to the fundus; and there press it against the uterine wall on all sides, so as to squeeze out the acid. Then withdraw it. Remove the cotton wool and the volsella, and then syringe the vagina with the sublimate solution. A good way of holding the wool is to wrap it round a slender piece of firewood having notches cut in it about an inch from the end. The wool can be tied on with string, tightly tied round it opposite the notches. It must be well tied, or there will be danger of its remaining in the uterine cavity. I prefer letting the forceps be damaged by the acid. They can easily be polished again.

This treatment does not invariably cure, but it does so in the majority of cases. Cases in which there is one single outgrowth are the most favourable. Those in which the growths are numerous and large are the most liable to relapse. Should relapse occur, the treatment must be repeated. In the diffuse form, when one curetting and cauterisation has proved insufficient, two or three repetitions will be followed by permanent cure.

If in a case with distinct polypoid outgrowths, speedy relapse and rapid growth of the neoplasm

indicates malignancy, the uterus should be removed without delay.

The disease I have just described is not endometritis, although it is often called endometritis. If there be with it inflammation, this is an accidental complication, not a regular part of the disease.

I shall now describe endometritis, properly so-called. In endometritis the lining of the uterus presents the macroscopical and microscopical characters of inflamed mucous membranes. It secretes pus; on section the tissue is seen to be infiltrated with leucocytes; later, there is organisation of these leucocytes into connective tissue; degeneration of glandular epithelium, and thickening of the walls of small arteries by hyaline connective tissue.

The causes of this disease are:

Puerperal Infection. In the puerperal state endometritis is likely to be produced if a bit of membrane or placenta remain in the uterus, and micro-organisms of a less virulent kind than those which produce blood-poisoning get to it. When this happens the bit of retained stuff decomposes, and the endometrium becomes inflamed. We do not yet know which are the micro-organisms that bring about this puerperal disease and no other. The symptoms of the endometritis are that the lochia are in excess; the flow remains bloody longer than it should do; when it ceases to be bloody it is still more profuse than it should be, and is first rusty, then purulent. There may be slight fever, and the lochia may stink; these two latter symptoms usually go together. The involution of the uterus is retarded, and therefore the uterus is bigger than at the period of involution it ought to be.

Course of Puerperal Endometritis.

(1) It may be treated and cured by removal of the retained stuff with forceps or curette, and irrigation of the uterus with sublimate solution, 1-2000.

(2) It may be spontaneously cured by expulsion of the retained stuff, and restoration of the endometrium to a healthy state by the action of leucocytes. This event, although possible, cannot be counted on.

(3) Natural cure, though it take place, may be

slow and incomplete, and discharge linger on for months after delivery.

(4) The inflammation may spread to the Fallopian tubes, and from them to the peritoneum and ovary, and thus chronic salpingo-oophoritis be the result.

I have described puerperal endometritis as due to retention of a bit of placenta or membranes, because these are the cases in which the mode of origin is clearest. But in many cases there is no evidence of anything having been retained. Puerperal endometritis may be produced without this favouring condition, but we do not know how, in such cases, it comes about.

Symptoms of Chronic Catarrhal Endometritis.

These are very slight. White or yellow discharge. Pruritus, if the discharge is irritating. Menstruation may be increased in quantity, but is not always. In most cases of leucorrhœa the discharge comes from the vagina, and can be cured by vaginal injections. In some the cervix is thick, granular, and inflamed, and furnishes some of the discharge. In these, treatment of the cervix and the vagina stops the discharge. In a few the discharge continues after the cervical erosion has been cured, and notwithstanding the use of vaginal astringent injections. If this is the case, you may infer that the discharge comes from the body of the uterus. It is only in this way, that is, by the failure of treatment applied to the lower part of the genital tract, that chronic catarrh of the body of the uterus can be found out.

Gonorrhœa may cause endometritis. As the patient already has a discharge from the vagina, the spread of the inflammation to the uterus causes no change in the symptoms. It may spread from the body of the uterus to the Fallopian tubes, and thence to the peritoneum. When this happens, there are no symptoms by which the extension of the disease can be detected until the peritoneum is reached. If such extension takes place at all, it takes place early. If the peritoneum suffers, it is usually within two months of the date of infection.

Gonorrhœal inflammation of the vagina may get well without treatment, although more slowly and imperfectly than if proper treatment be used; and so, I doubt not, may gonorrhœal endometritis. If it does not, the only symptom it causes is discharge, and the same rule of treatment applies here

as in endometritis of puerperal origin. If the discharge continues in spite of treatment of the cervix and vagina, you may conclude that it comes from the body of the uterus.

A *dead fibroid*, whether disintegrating or not, may set up purulent endometritis. If micro-organisms get into the uterus, they find in the dead tissue a soil suitable for their growth. *Cancer* sometimes does so, in like manner. Other rarer tumours, such as *sarcomata* or *adenomata*, may also do it. In most cases this endometritis is a disease secondary in importance to the condition which caused it. But it may spread along the tubes, and lead to peritonitis.

There are alleged causes of endometritis, which a French writer* has grouped together under the happy term "*banal*." This word, according to the dictionary, means trite, commonplace, hackneyed; and in matters medical this often means accepted and copied from book to book without inquiry. Some of these "*banal*" causes probably do cause endometritis, although there is no scientific proof of it in the case of any of them.

Among these banal causes are (1) *Suppression of menses*. If any harm comes from this it probably is endometritis. But the menses are often suppressed without harm; and we do not know in what cases it does harm, or how, or why. (2) *Retroversion and retroflexion*. In most cases the changes in the shape and position of the uterus causes no trouble. In about ten per cent. the circulation is interfered with, and morbid effects follow, endometritis possibly being one; but the nature of the changes in the uterus has never been demonstrated. (3) *Anteflexion*. (4) *Congenital stenosis*. Neither of these peculiarities produce any morbid effect. (5) *Overloading of the bowel*. This makes people feel ill; but I know of no *à priori* or other reason for supposing that it causes endometritis. (6) *Over-exertion during menstruation*; such as from riding, driving, dancing, treadle-work, as with a sewing-machine or bicycle. Many women do these things while they are menstruating as well as at other times, and without harm. Continued to the point of fatigue they are injurious, whether the patient is menstruating or whether she is not. I know of no evidence that endometritis is the special form of disease apt to be set up;

but as such causes may produce uterine hæmorrhage, I think it possible that they may cause endometritis. (7) *Excessive sexual activity*. This cause leads to increase flow of blood to the generative organs, and such hyperæmia may perhaps go on to the production of endometritis; but nature commonly prevents this by hæmorrhage, which relieves the hyperæmia.

Endometritis in Febrile Diseases. In acute febrile diseases there is sometimes endometritis. I have seen the uterus full of pus in a young girl dead of enteric fever. In cholera, enteric fever, measles, scarlet fever, and smallpox, hæmorrhagic endometritis—called also "*pseudo-menstruation*"—has been described. Such endometritis is at the time subordinate in importance to the constitutional disease which produces it. The state of the patient forbids local treatment unless absolutely necessary, and the effects of the endometritis are not serious enough to urgently require treatment. I have never been called upon to treat such endometritis. Chronic endometritis following these diseases is rare; but I have known increased menstrual hæmorrhage and leucorrhœa follow measles. As these febrile diseases, taken collectively, are common, it is probable that such endometritis usually ends in recovery when the disease causing it has done so.

Symptoms of Endometritis. The symptoms of genuine endometritis, whether gonorrhœal or of banal origin, are very slight. There is little or no pain. The discharge from the vagina is not augmented to a degree which enables you to assert that catarrh has spread to the endometrium. The diagnosis is made by the physical signs. The cervical canal is dilated, so that a Playfair's probe with cotton wool wrapped round it passes to the fundus easily. Round the os externum the mucous membrane is red, and has lost its smooth and shiny appearance. There is a rusty, bloody, or purulent discharge issuing from—not merely lying about—the canal. Passage of a sound causes bleeding.

Treatment of Acute Endometritis. Like all inflammations, endometritis will get well if its cause be removed, and the patient put under favourable conditions. The one serious consequence of endometritis is the extension of the disease to the peritoneum; if this be averted the disease is a trifle. Keep the patient at rest; this is most effectively attained by ordering her to bed. Keep the vagina

* Doléris.

clean, and lessen inflammation in it by antiseptic and sedative or astringent douches; a saturated solution of boric acid if the vagina be sore or the discharge irritating; chloride of zinc, $\frac{1}{4}$, $\frac{3}{8}$, or $\frac{1}{2}$ a grain to the ounce, if the discharge be copious. This will prevent the addition of fresh germs to those with which the leucocytes are already battling. Under such treatment most cases will get well in a week or two.

So-called Atrophic Endometritis. In some cases of hæmorrhage from the uterus in women near the menopause, when, after dilatation of the cervix, the uterine cavity is explored, no morbid change can be detected by the finger; and if the curette is used, either no solid shreds at all are detached, or only very few and small pieces. Microscopic examination of the little bits removed shows a firm stroma of spindle-shaped elements; few glands, and those with narrow lumen and small epithelial cells; closed arteries with thick walls; here and there groups of broken-up nuclei taken to indicate places where glands had been. In some uteri examined after removal from the body during life or after death, the endometrium has been found very thin, presenting histological changes like those described above, with the addition that the columnar epithelium may be found changed into squamous, and in some places small, cysts, formed out of blocked and dilated glands, exist.

German writers call such cases *atrophic endometritis*. They think it is a final stage of endometritis which leads to atrophy. The only reason that I can find for this belief is that sometimes in the same case one scraping will bring away pieces showing signs of hyperplasia and catarrhal inflammation, another scraping nothing, or only pieces showing the signs of atrophy; and it is inferred that the scraping which brought away nothing, or a little piece showing signs of atrophy, was made at a place where the morbid change had gone on to atrophy. Now, unless we know exactly at what period of the menstrual cycle the examination was made, the fact of atrophy is not proved; for the mucosa is always thin at a certain time in the month. When patients are losing blood irregularly they often lose reckoning of when menstruation ought to come on. But passing by this point, I know of no evidence that this atrophic condition is a result of inflammation. It is true that evidence is very difficult to

get. But that is no reason for taking a theory to be fact without evidence. The term "*atrophic endometritis*" expresses a theory.

In these cases there is little or no pain. Leucorrhœa is not great; there is not the pink watery discharge of adenoma, nor the purulent discharge of endometritis. The patient's trouble is that she loses too much, and loses too often. The bleeding makes the patient anæmic for a few days, but it is seldom enough to cause a high degree of anæmia. The result of treatment is unsatisfactory. Scraping and cauterizing stop the bleeding for a few weeks or months, and then it returns. Ergot does no good. The only treatment is repetition of local applications to the endometrium as often as the hæmorrhage recurs to a weakening amount. After, by exploration you have made sure that there is no intra-uterine growth, it will be enough to treat the hæmorrhage by a simpler method of application, such as by Playfair's probes, or the injection of small quantities of a styptic solution into the uterine cavity. The safest is the tincture of hamamelis.

These cases usually occur in women near the menopause. This fact, the insignificance of the appreciable local changes, and the non-success, or very transitory success of local treatment, makes me think that the cause of the hæmorrhage is probably vascular degeneration. Degeneration of small blood-vessels occurs in old age; and in a woman at the climacteric the uterus has reached old age. Mr. Hutchinson has suggested that such hæmorrhage is sometimes due to gout. But the signs by which to tell whether a patient is, or is not gouty are so indefinite that it is very difficult to test this theory.

Alumol in Gonorrhœa.—Chotzen (*Journ. de Médecine de Paris*) holds that alumol, 1 per cent., destroys gonococci without increasing inflammation. Bacteriological examination of the discharge shows beyond cavil the inhibiting action of the alumol upon the gonococci. For anterior urethritis a 1 or 2 per cent. solution (3iss) is employed at first six times a day, later three times a day. When the gonococci disappear, the solution is reduced to $\frac{1}{4}$ — $\frac{1}{2}$ per cent. In posterior urethritis a solution of from 1 to 5 per cent. is instilled, or alumol ichthyolate is employed in 2 to 10 per cent. In case of urethral or cervical gonorrhœa in women, injection of alumol ichthyolate is employed, also alumol bougies.

TWO LECTURES ON THE TREATMENT OF DIPHTHERIA BY ANTITOXIC SERUM.

LECTURE I.

Delivered at University College Hospital.

BY

SIDNEY MARTIN, M.D., F.R.S., F.R.C.P.,

Assistant-Physician to the Hospital.

I PROPOSE to-day to discuss some of the recent points concerning the pathology of diphtheria, and its treatment by antitoxin. The fundamental fact, the basis of this method of treatment, is that diphtheria, as we now know it, is due to the bacillus known as the bacillus diphtheriæ producing its poisons in the membrane, which are then absorbed in the body. There are two kinds of treatment, local and general. The local treatment has for its object the detachment of the membrane, and the killing of the bacillus present in it. The general treatment has for its object the sustaining of the strength of the patient, the counteracting of the poisons which are produced in the membrane, and the prevention of the growth of the bacillus.

The method of counteracting the poisons and the prevention of the growth of the bacillus are the outcome of modern research. In the general treatment we seek for an antagonist to the poison, and in local treatment our aim is to destroy the bacillus.

Some cases of diphtheria will get well on local treatment only, but you never know when such patients are going to die. In the faucial and pharyngeal cases the patient may, as regards his general condition, be going on well; the membrane may disappear, and yet the patient may die suddenly from syncope. Heart failure was, therefore, one of the causes of death in the pre-antitoxin period; more died of paralysis, and a smaller number of suppression of urine, and a still smaller number died of inflammation of the kidneys.

In laryngeal cases the cause of death was, as a

rule, asphyxia, sometimes broncho-pneumonia; and indeed, in most pre-antitoxin cases, one found broncho-pneumonia. In both classes of cases death might occur from pus infection; some dying from sapræmia, resulting from decomposition in the membrane. Lastly, both classes of cases sometimes ended fatally in acute bronchitis.

The first point in the modern theory to discuss is the nature of the toxins produced. A tabular statement of these can be arranged in columns; in the first is the bacillus diphtheriæ, in the next column is the ferment or toxin, and in a further column are certain products, albumoses and an organic acid.

Bacillus diphtheriæ	Giving rise to 1. Toxin or ferment (Roux and Yersin's "diastase")	2. Albumoses and an organic acid, digestive products.
---------------------	--	---

In the body itself the poison is found in the blood and the organs, especially in the spleen. A small quantity of albumoses are found in the membrane, but it is in the spleen where these albumoses accumulate. These bodies are prepared artificially from the diphtheria bacillus, and they have the same action as those produced from the body, thus completing the proof that the bacillus diphtheriæ is the cause of diphtheria.

The toxin is made by growing the bacillus in the ordinary broth made from meat and rendered alkaline, 0.5 per cent. sodium chloride and 2 per cent. commercial peptone being added. It is a remarkable fact what degree of alkalinity the bacillus diphtheriæ can stand. It grows best in a strong alkaline medium, which gives a blue colour to litmus and a bright pink with phenol-phthalein. In this solution albumoses are not produced, but if alkali-albumin is substituted for the peptone the toxin alone is not produced, but the albumen is gradually digested, albumoses being formed. The bacillus is allowed to grow in the alkaline peptone broth for a month, when a copious deposit of the bacillus is found at the bottom of the flask; the more air allowed to enter into the bottle the more toxin is produced. The bacillus can be removed by filtering, or killed by adding 0.5 per cent. of carbolic acid.

The activity of this poisonous solution is remarkable; $\frac{1}{10}$ to $\frac{1}{20}$ c.c. will kill a guinea-pig,

200—300 grammes in weight, in forty-eight hours. When you consider that the amount of solid matter in this amount of liquid (after deducting the quantity of salts and peptone) is imponderable, some thousandths of a milligramme, you will see in what infinitesimal doses this toxin acts. If you inject subcutaneously as little as 0.3 c.c. of the toxin it kills a rabbit in about twenty-four hours. If you give smaller doses to rabbits and guinea-pigs, wasting, diarrhoea occasionally, and paralysis occur; and post mortem you find fatty degeneration of the heart and nerve degeneration. Fever of a more or less severe nature appears after a few days from the slow action of the toxin, the effects not being seen at first. One of the new facts about this toxin which experiment has shown is that it acts only after a period of incubation.

Here is a non-living substance producing or reproducing the symptoms caused by a living organism, and reproducing the symptoms after an incubation period; such a substance may be called provisionally a ferment.

If the alkali-albumin broth containing albumoses is used for injection after filtration, it is found that to kill a guinea-pig large doses must be used. This produces, first of all, fever, and then a gradual series of symptoms exactly reproducing the features of a case of diphtheria in a human being,—depression, wasting, diarrhoea occasionally, if the dose is large, and paralysis; post mortem, fatty heart and nerve degeneration being found. These are the same symptoms as from the toxin, but fever is produced, and much larger doses are required.

The antitoxin is formed by injecting the toxin into a healthy horse; a strong toxin is needed, one that kills a guinea-pig with $\frac{1}{10}$ c.c. At the commencement very small doses are injected, sometimes $\frac{1}{8}$ c.c., and sometimes 1 c.c.; larger doses to begin with may produce serious results. The horse reacts to the toxin with some general disturbance, and perhaps a little fever afterwards. On the horse recovering another dose is given; and if there is no reaction, the dose is gradually increased to 100 c.c., or more, of toxin of the same strength. As the injection proceeds, the blood serum, after a few weeks, is found to contain an antitoxic substance. At the end of some months this serum contains a large quantity of the substance. The antitoxic serum in use is obtained by bleeding the horse when the blood is at its highest antitoxic

strength. Eight litres of blood are drawn into a sterilized vessel, and the serum removed after the formation of the clot. Another eight litres can be taken from the same horse in a month, and this can be repeated for some time. The horse, if well looked after, keeps in excellent condition.

The strength of the serum is of great importance in the practical application of antitoxin. With a toxin, $\frac{1}{10}$ c.c. of which kills a guinea-pig weighing 200—300 grammes in about 48 hours, if you want to test the serum you take 1 c.c. of the toxin (*i.e.*, ten times the lethal dose), and add to that different proportions of the antitoxic serum. For example, with one animal experimented on, $\frac{1}{100}$ c.c. of the antitoxic serum is added, and with another guinea-pig $\frac{1}{10}$ c.c. is added, to another $\frac{1}{5}$ c.c. is added, and it is observed whether the animal survives. If the animal survives in the first case, it is said that one $\frac{1}{100}$ c.c. of antitoxic serum is capable of counteracting ten lethal doses of the toxin.

The unit which is taken for measurement is ten times the amount of serum requisite to counteract ten lethal doses of the toxin. Thus, if $\frac{1}{100}$ c.c. of the serum counteracts ten lethal doses, $\frac{1}{10}$ c.c. multiplied by ten contains one normal unit, therefore 1 c.c. will contain ten normal units, 20 c.c. will contain 200 normal units.

Nos. 1, 2, and 3 of Behring's antitoxin contain respectively 600, 1000, and 1500 normal units.

The practical point is, that the stronger serum you use the better for the patient, and one must be guarded in drawing deductions about the treatment of diphtheria by antitoxin, unless the strength of the serum used is known and of a certain degree. If weak antitoxin be used and bad results follow, the fault must not be imputed to the antitoxin treatment.

Antitoxin is preserved in sterilized bottles, and it is kept by adding an antiseptic, such as carbolic acid, tricresol, or camphor.

It may be asked, what does the antitoxin actually do? Part of its action I have already referred to; it counteracts the effects of the toxin, but it does more than that. It is necessary to study the effect of the antitoxin not only on the toxin itself, but also on the toxic bodies found in the tissues of the diphtheritic patients—these bodies being the albumoses. First of all, the antitoxin mixed with toxin counteracts the effects of the poison when injected into animals. It is noticed that after a time some

of the animals live and some die ; even though the control animal die under forty-eight hours, some of the antitoxin animals die in from twenty to eighty days. These animals present in the course of their illness the symptoms noted in diphtheritic patients. Wasting occurs, and after death the characteristic nerve degeneration and fatty heart are found, showing that though the animal survived the immediate effect of the toxin, the antitoxin did not act on all the effects of the toxin, and degenerative changes occurred, ending in death. The animals that receive a larger dose of antitoxin than those just referred to, remain perfectly well and gain in weight.

If the antitoxin is injected a few hours after a lethal dose of toxin has been given, there is great difficulty in saving an animal. Very large doses must be given, and even then you only sometimes postpone the fatal end. To counteract all the effects of the toxin when present in the body, large doses of antitoxin must therefore be given. Even though the animal may live, get fat, and be apparently well, some nerve degeneration may still be found after it is killed, showing that the test in common use for the strength of antitoxin is only an approximate one.

In the post mortem of surviving animals which have received both toxin and antitoxin, one important practical point is fully borne out—no fatty degeneration is found in the heart. The antitoxin therefore completely counteracts the effects of the toxin on the heart. The post mortem on animals which survive shows slight nerve degeneration, much less, I may say, than in animals not treated by antitoxin, but does not show any fatty degeneration of the heart. It has been observed that though a horse may possess a large quantity of antitoxin in its blood, still on the injection of albumoses the animal shows signs of fever. Antitoxic serum has only a slight effect on fever. As regards the effect of injecting antitoxin after albumoses have been injected, the point has not yet been worked out, but the investigation, as far as it has gone, appears to show that the antitoxin is capable of counteracting many of the effects of the albumoses if injected after the albumoses.

The last fact about antitoxin is that it confers immunity. If a guinea-pig receive an infinitesimal dose of antitoxic serum, and twenty-four hours afterwards a lethal dose of the bacillus, it will

survive, showing that immunity from a virulent culture of the bacillus has been conferred. In the treatment of the human sufferer from diphtheria this point is of the greatest importance.

In diphtheria, the symptoms are gradual in their onset, and in the course of the disease the toxin at different intervals is passed into the system not in one big dose, which may, however, occur in some cases, but in small doses ; following on this is the gradual accumulation of the albumoses, especially in the spleen, the physiological effects of which may continue after the bacillus has disappeared from the throat.

This gradual and repeated entrance of the toxin into the body, and the gradual accumulation of the albumoses are of prime importance in considering the action of the antitoxin. The membrane itself increases up to a certain point, owing to the growth of the bacillus. If, therefore, we have a substance which will counteract the effects of the toxin, and the albumoses, and will confer immunity, so that the bacillus ceases to grow, then we have a powerful agent in counteracting the disease. I may add here that the effect of this immunity is well seen in the membrane if the antitoxin be given in large doses, the membrane not only ceasing to spread, but rapidly disappearing. The bacillus diphtheriæ may live in the throat for seven or eight weeks, or longer, after the membrane has disappeared, and, though it remains as virulent as ever, it yet produces no lesion. This must be because the tissues of the body have been rendered immune to the bacillus, being in such a condition that it cannot form a false membrane.

The treatment of diphtheritic patients is not quite on the same lines as the experiments in the laboratory. The patients have the poisons already in their bodies, and in their spleens albumoses have accumulated to a greater or less extent. Therefore, to bring the patient into a condition of immunity, the poison already in the tissues, and any poison which may form after the patient has been seen must be counteracted. The earlier the treatment is begun the less is the amount of poison present, and the more rapidly is immunity produced.

Cases of diphtheria present themselves under different characters, and may be divided into doubtful cases, cases with membrane, laryngeal cases, and lastly cases following scarlet fever, or cases with complications. These four classes are

the chief practical divisions to be dealt with. The doubtful cases are those with a history of infection and a doubtful throat, the throat being red, congested, and swollen, with perhaps a doubtful membrane over the fauces consisting of a thin layer of pultaceous mucus. Membranous cases with what is called a "leather throat," with a leathery membrane over the soft palate, need no special description. The procedure essential to follow in all these classes is, first of all, isolation, treating even the doubtful ones as cases of diphtheria; the second point is to inject antitoxin, not waiting for a diagnosis before injecting; the third point is to make a cultivation from the throat, which will decide the diagnosis in twenty-four hours.

When a patient is first seized with diphtheria it has to be decided whether it is a severe case or not. What is meant by a severe case? To decide this, first of all look to see if there is a large amount of membrane; secondly, look for the signs of the patient being poisoned. Many have a poisoned look, with an earthy and pallid complexion and great bodily depression. Another sign of poisoning is the early onset of paralysis. In this matter the loss of knee-jerks must guide our judgment; the earlier the knee-jerks are lost the more deeply is the patient under the influence of the poison. Another sign also in connection with the knee-jerks occurring in some cases is great exaggeration of this reflex. Lastly, you have, as a sign of poisoning, great diminution of urine, with early albuminuria. Fever is a doubtful point in diphtheria in judging of the severity. In many of the cases that end fatally there is only very slight fever; nevertheless, prolonged and high fever is a sign of the severity of the case.

The association of the streptococcus with the bacillus diphtheriæ in the throat is very serious. It is not always right to give a bad prognosis when streptococci are found with the diphtheria bacillus in the throat; but the presence of a large amount of streptococcus is serious.

The last practical point as regards the severity of any case of diphtheria is its association with other diseases, such as scarlet fever. The patient may die from acute bronchitis, and diphtheria may follow acute bronchitis, or it may occur in a child which has had scarlet fever followed by Bright's disease.

CLINICAL NOTES.

WITH DR. CHEADLE
IN THE WARDS OF ST. MARY'S HOSPITAL,
31st March, 1896.*

Influenza.

THIS was a case of a boy, æt. 6, who was admitted into the hospital complaining of cough and headache. There was a history of the illness coming on nine days before his admission. When he came in there were signs of general bronchitis. There was no dulness, but there were coarse crepitations, particularly loud at certain places, which appeared to vary in their position from time to time. There was abdominal distension with diarrhoea, but the stools were of a dark colour. The prostration was well marked. The question was discussed as to the probability of typhoid, but the patient's temperature came down to below normal in a very short time. The case was clearly one of some acute contagious catarrh with extreme prostration and slight broncho-pneumonia. On examining the lungs at the back intense fine moist crepitations were heard, but in front the lungs were clear, except at the lower border of the left lung. This case was benefited by the exhibition of digitalis, and the circulation underwent rapid improvement. The patient was a good example of the kind of influenza which is now so much about; the great feature of which is the persistent character of the bronchial catarrh, often lasting for weeks, and accompanied by extreme prostration.

Rheumatic Fever, Complicating Chronic Endocarditis.

This was a case with a well-marked double mitral murmur. There was a remarkably distinctly heard presystolic bruit, and there was considerable hypertrophy of the heart. The history of rheumatic fever only dated back three months, and if the mitral murmurs had been soft and blowing it would perhaps have indicated that the mischief was recent. The harsh character of the murmur, the hypertrophy of the heart, and the

* We are indebted to Dr. Cheadle's House Physician, Dr. Lane, for his courteous assistance in regard to this contribution.

very recent history of rheumatic fever pointed to the fact that, in this case, the endocarditis had undoubtedly existed before the attack of rheumatic fever.

Early Typhoid, or Relapse.

This patient was a boy, æt. 8, who was admitted into the hospital with abdominal distension, and a history of a continued high temperature—about 104° . There was a history of two or three weeks' *malaise*, but no diarrhoea. On admission the only symptoms were fever and general *malaise*, the abdomen being slightly distended; there were no spots; the spleen could possibly be felt. The definite symptoms in this case seem to have commenced about a month ago, and the question arose, was this to be considered a relapse, or was it one of those instances of the prodromal stage of typhoid lasting for a very long time; sometimes the prodromal stage is observed to continue in this manner. This child was running about, apparently only slightly indisposed, till admitted into this hospital; and it is well known that adult cases of typhoid occur answering in some respects to the condition we have here, and known as ambulatory typhoids; by the time, however, weakness has compelled them to lay by, the amount of reserve strength in their systems is generally too little to prevent serious results.

Last Stage of Mitral Disease, with Ascites.

This was an instance of the result of mitral disease in a young woman æt. 17. She had been in the hospital before, a few months ago, for her shortness of breath. Her heart disease had been treated, and she was discharged; but she had now returned complaining of pain in the abdomen, sickness, and increasing shortness of breath. Upon examination there was found mitral stenosis and regurgitation with tricuspid mischief; the abdomen was greatly distended with fluid, and dull all over. The patient was tapped at once, because of the dyspnoea. Such cases as these form a distinct group in cardiac dropsies. At a certain stage, either because there is peritonitis, or because the liver has become fibrous, ascites occurs; sometimes by itself, sometimes with general dropsical symptoms. In this case there is only a very slight œdema about the feet in addition to the ascites. In an earlier stage than this several instances

have occurred in which after paracentesis recovery has not only taken place at the time, but the ascites has not returned for several years.

In discussing this case an instance was quoted in which a patient recently in hospital had been tapped for ascites forty years ago by Dr. Page of St. George's. The man's abdominal walls were found to be thickened and hard, with a large deep-seated mass in the abdominal cavity, probably from old tubercular inflammation of the peritoneum. The opportunity for the subsequent examination was afforded by the patient attending St. Mary's for some minor ailment. The tapping performed forty years ago for ascites completely cured him of the dropsy, but the nature of the original ailment was obscure.

Alcoholic Cirrhosis, Large Hard Liver, Catarrhal Jaundice, Perihepatic Inflammation.

This patient came into the hospital complaining of jaundice. On examination the liver was found reaching to the umbilicus, and was hard and granular to the touch. There was no ascites. Three days after admission she developed delirium tremens. Morphia did not induce sleep, which was procured in this case by hyoscin. Ascites was first noticed fourteen days ago, and has slowly increased. She now complained of pain in the back and at the side, and upon examination her lungs were found clear. The tenderness over the liver, its enlarged condition, and the position of the pains, indicated perihepatitis. A plaster of mercurial ointment was ordered to be applied on the tender hepatic region as the most likely means of subduing the perihepatic inflammation and relieving the condition. Instructions were also given that warm fomentations were to be applied to the abdomen.

Syphilitic Affection of the Larynx.

This was a case of a middle-aged woman who came to the hospital complaining of what she called bronchitis and asthma. On examination of her lungs nothing could be made out. There was however evident dyspnoea. Her cough got no worse and was not of a spasmodic character, and there was no secretion or expectoration. The conclusion was arrived at that there was no bronchitis and no asthma. The fourteenth day after

her admission localised ronchi were noticed on auscultation; but, as well, there was a general sound all over the chest, which seemed to be conveyed from the larynx. An examination with the laryngoscope disclosed only catarrh. It was decided that the affection must be of a laryngeal or tracheal origin, but nothing could be found in the throat to substantiate this idea, except an elastic rounded body about the size of a chestnut lying close to the trachea below the larynx on the right side—apparently an enlarged gland. It was freely moveable, and was noticed to move on deglutition. The distress in breathing and the loud wheezing were judged to be connected with either malignant or syphilitic disease of the larynx, of which the enlarged gland was an indication. There was no history to suggest specific origin of this affection, but it was thought advisable to give the patient iodide of potassium, with the result that the laryngeal trouble was quickly relieved, although the size of the cervical gland has not decreased. The dyspnoea and the wheezing sound have quite gone. The patient is perfectly free from any respiratory difficulty, and is going out apparently well. The iodide will, however, be steadily continued for some weeks longer.

Gastric Ulcer.

This patient was a young girl who had just been admitted into the hospital because the day before she had vomited up blood. She has always for the last two years had pain about ten minutes after taking food, and after solid food has vomited. On the occasion of vomiting blood she first fainted, and then brought up the blood. The explanation of this is that the blood pours into the stomach, causing faintness and subsequent vomiting. Upon examination it was noticed that there was some distension of the stomach, and much pain was complained of below the left costal margin, great care being taken of course to make the examination as gently as possible. The main points were then commented on, and the order of the pathological events, viz., first the anæmia, then the enfeebled circulation, the possible thrombosis, and lastly the erosion. In the treatment of this class of case one important point was insisted upon, and that was that not only must feeding be absolutely restricted to the usual nutritive enemata—no fluid even being allowed by the mouth; but it

is essential also to provide an ample supply of fluid by the bowel—a matter of the first importance in the treatment of these cases, although frequently overlooked or neglected. An injection of half a pint to a pint of simple water to be retained should be given twice a day, in addition to the regular enemata. This point was very clearly brought home by the history of a case of a woman who was being fed *per rectum*, because of her incessant vomiting. She received the ordinary nutrient enemata, but in spite of this she became pinched, cold, collapsed, and shrunk to an extraordinary degree, so that she looked very much like one of those cases seen in children when they are drained of fluid by the incessant vomiting and diarrhoea of severe gastro-enteritis. She was in a state of extreme danger from collapse. A large injection of fluid was administered to her by the bowel: this was retained, and no doubt at once sucked up by the absorbents as by a sponge, and the woman rapidly recovered. Provided a patient has plenty of water, comparatively little other nourishment is necessary, and the practical importance of remembering the necessity of providing that supply was strongly insisted upon.

Hyperpyrexia without Definite Symptoms, probably Rheumatic.

This patient was a girl who came in about a fortnight ago for pains in the legs and feet, with a history of a six days' illness. On admission her temperature was 100, and in two days was 104, since when it has very gradually fallen. Her tongue was coated on admission, and there were no definite symptoms pointing to rheumatic fever. No satisfactory cause could be found for the persistent high temperature. Quinine was at first administered, but had little effect; salicylates, however, were more efficient. The most probable hypothesis put forward in explanation of this case was that she might have been on the verge of hyperpyrexia, which would point to the rheumatic nature of the affection. On admission it was noted that the patient had a stupid look, almost amounting to a stuporose condition, and seemed otherwise affected as regards her intelligence. All this has passed off, and the patient is now bright. It is possible that the case may have been one of influenza.

Rheumatic Chorea in a Woman, æt. 27.

The curious points about this case were that the choreic movements were almost entirely confined to the neck, and that the age of the patient was seven and twenty, very late for the supervention of a pure rheumatic chorea. It was not known what had brought on this present attack, but the woman had had rheumatic fever, arthritis in the knees and hands, and three attacks of chorea when a girl. The family history was of a strongly rheumatic character. There was no question in this case of pregnancy. The unusual features were the implication of the neck and head, the mature age of the patient, and the absence of any immediate exciting cause, although it was clearly one of a series of rheumatic attacks, numbering four of chorea alone in addition to arthritis, three of which had occurred about the age of 14.

Case of Suspected Addison's Disease.

The patient was a woman of about 40, who has been under observation for six months, and the broad features of the case which presented themselves for diagnosis were constant vomiting, pain in the back, tenderness in the region of the right kidney, and slight general bronzing. With regard to pernicious anæmia, it was observed on examining the blood that the red corpuscles were few and altered in shape. The dyspeptic trouble was so severe that the patient preferred to have nutrient enemata rather than take food by the mouth. In regard to the pigmentation, there was no particular increase in the general bronzing at the thighs, on the mucous membrane, or at any points of pressure. The fact was also elicited that the bronzing had come on within the last six months. In respect to the feeding of this patient, the use of eggs in enemata was not recommended, and the practice advocated of using two ounces of milk and half an ounce of beef jelly pancreatised by the addition of liq. pancreaticus.

Sickness from Digitalis.

In the next case, which was one of cardiac trouble, the administration of digitalis by the mouth had caused distressing sickness, and the only way in which the drug was borne in this case was hyperdermically. Another instance was mentioned, also of heart affection, in which digitalis

caused sickness, whether given by mouth, or rectum, or hypodermically; and its use had to be abandoned altogether.

A Doubtful Case of Malignant Disease, or Ulcerative Endocarditis.

This case was a woman, æt. 47, admitted into the hospital for slight joint pains and a high temperature. Though she had dyspnoea, there was no physical sign of morbus cordis, or of lung affection; and why the temperature had been so high was a matter of uncertainty. For the first time in the course of this illness a pleuritic rub in the region of the left axilla was noticed on that day (March 31st). The patient had been in bed for three weeks, and this was the first time any sign of pleurisy had been observed. At the back of the left lung there was bronchial breathing, a well-marked rub, and a want of resonance at the base. One point which was noticed was the localization of the pleurisy to one lung. There was no œdema. There was no pain over the region of the spleen, and no signs could be found pointing to ulcerative endocarditis, and no positive indication that malignant disease was the cause of the trouble. The diagnosis in this case was reserved until the patient had been under further observation.

Syphilitic Affection of the Liver.

This case was a boy, æt. 15, who came to the hospital on account of distension of the belly and pain in the epigastric region. There was no family history to be obtained. On admission hard nodular masses of growth were observed along the anterior border of the liver or connected with the omentum. These masses were first noticed six months ago. When he first came in the signs of keratitis were marked, and with the ophthalmoscope this indication could be now confirmed. The pain was constant, and distressed the boy much. He was treated with biniodide of mercury, but not much improvement was noticed. He was then given iodide of potassium, and there was at once a marked change for the better; in a very short time the hard masses became softer and lessened in size, and the patient was relieved of his pain, his moaning and groaning stopped, and he was happy and comfortable. Although this case was in all probability one of tertiary syphilis,

apparently of congenital origin, still the possibility was pointed out of it having occurred from a primary infection; and a case was quoted of an entire family becoming infected from the mother's lips. The history of this boy had in it nothing suggestive of any specific character, except the interstitial keratitis, there being no record of snuffles, rash, etc., and the theory of the gummatous nature of the swelling was at first discussed.

Stricture of the Œsophagus.

This man had been under observation since Christmas, and came into the hospital because he was suddenly seized with inability to swallow food. On being questioned this appeared to have been an acute exacerbation of some slight difficulty he had been noticing for some little time, he having gradually been choosing liquid food because of the greater ease in swallowing. The inability to swallow even liquid food came on quite suddenly, and the case furnished no evidence of any pouching. The food was immediately returned on its introduction, being vomited, without any retching, through his mouth and nose in a kind of puking manner, there being a marked absence of any kind of spasmodic effort. The case was carefully examined with the view to exclude aneurysm. No cause was to be assigned in this particular case, and the existence of prolonged dyspepsia was discussed as one of the possible causes by setting up malignant stricture of the Œsophagus. Another possible cause was syphilis, but the absence of any history pointing in that way and the failure of specific treatment to give any relief indicated that in this case it was in all probability not the cause. Injury as a cause was also excluded after a careful discussion of the symptoms and signs.

Pyloric Cancer.

This patient came to the hospital four weeks ago complaining of hæmatemesis, which appears to have been the first symptom noticed in his case. Upon examination tenderness was noted in the epigastrium, and deep down under the left costal margin a mass was to be felt. This had much increased, and now there was a hard dense mass very fixed, stretching across the epigastrium. The diagnosis of this case was discussed, and the malignant character of the growth was pointed out.

Old Contracting Empyema simulating Phthisis.

This was a male patient, æt. 14½, who had been sent in from an institution because of his hectic temperature. There was a history of enteric fever in December, complicated with pneumonia and pleurisy. On admission the right side of the chest was an inch smaller in circumference than the left. There was dulness in the right axilla in its lower part, and coarse crepitations could be heard all over the right side of the chest. The resonance was fair except in the region of the lower part of the anterior axillary line. A week after admission the lad's condition became much worse, and a needle was inserted into the chest at the anterior axillary line over the dulness, and about two ounces of pus were withdrawn. It was pointed out that the interesting part to be noticed in these cases was that you have all the signs of phthisis, and that they are very often mistaken for phthisical cases.

Dr. Cheadle stated that he, many years ago, thus mistook the first case of old contracting empyema he saw, and demonstrated it repeatedly as a case of phthisis. He was only induced to relinquish that opinion by the empyema pointing.

Probable Sarcoma of the Omentum.

This case was a lad about 13, who was admitted on 12th February last because of vomiting, with abdominal pain and enlargement. Upon admission a mass was noticed in the region of the umbilicus, reaching upwards towards the epigastrium and downwards towards the pubes, and almost into each flank. The skin was involved specially in the region of the umbilicus, where there was a slight discharge. This mass was partly resonant. For three weeks iodide of potassium was tried, but no good resulted. The mass had enlarged since his coming in, and the lad was getting much thinner. The left leg was markedly cedematous. The question of tubercular mischief was discussed, and it was pointed out that the rapid development of the symptoms and signs pointed rather to sarcoma as the probable diagnosis.

Parenchymatous Inflammation of the Kidney.

This patient was a youth admitted three weeks ago because of the oedema of his feet. He had

been in the hospital once before, during last September, when he came complaining of swollen legs. Upon admission three weeks ago there was one third albumen in his urine, in which also there was blood. There was no high tension, and the heart was normal, except for a slight dilatation. The patient now passes 48 ounces of urine daily, which is about the normal amount, with specific gravity 1018, and therefore presumedly it contains the normal amount of urea; but directions were given that the amount of urea should be definitely calculated. Upon discussing the treatment of this class of case the fact that raw meat-juice is supposed to have been the cause of this lad's improvement was commented on, and further it was shown that it was a mistake to restrict debilitated patients of this kind to a diet of milk and farinaceous food alone. A patient in this condition has been very heavily poisoned and reduced to a very anæmic state; and if the pulse is not of high tension and the anæmia is severe, and further, if the amount of urea passed is two thirds of the normal amount, there is no harm, but often enormous benefit in giving half an ounce of raw meat-juice twice a day.

Fæcal Accumulation, sent to the Hospital as a Case of Acute Intestinal Obstruction.

This was a case of a young man who was sent in complaining of pain in the stomach and abdominal distension, tenderness on the left side over the sigmoid flexure, and stoppage of the bowels. His history was that he had been operated on in last September for a perityphlitic abscess, and since then he had only been troubled with costiveness until about a week ago, when the present illness began. On being questioned this patient connected his illness with some under-done pork he had eaten, and the possibility of his having been poisoned in some way was put forward. The question of adhesions from the old perityphlitic trouble was also discussed. After the man had entered the hospital he had two stools, and it was shown that the hospital was not responsible for the somewhat heroic dose of purgative medicine that this man had taken before coming in. It was also further pointed out that in spite of the man's appearance of illness he was now free from pain, and could lie comfortably in any position.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, North-West London Hospital,
March 18th, 1896.

DR. CAGNEY in the Chair.

Continued from page 376.

Mr. JACKSON CLARKE showed a case of Paget's Disease of the Breast.

Paget's disease begins as a small patch of erythema, soon covered by crusts, situated on the summit of the nipple, and extends progressively, but very slowly, to the areola and skin. After lasting from two to six years, or even as long as twenty years, it terminates in cancer of the breast. In the present case the affection is of the most typical form, that is to say, it is not hidden by scales and crusts. The region of the nipple and of the skin around for a radius of about two inches is of a red colour, in parts tending to rose, but for the greater part of a claret colour. As far as the lesion is concerned the most characteristic feature is the sharply-defined border. The patient is a woman of 43. She first noticed the beginning of the affection seven years ago as a scurfy patch on the surface representing the nipple, which in both breasts has never had any elevation, and had prevented the patient from suckling her six children. The aspect of the lesion varies at different times. Slight excoriations appear, giving rise to a serous discharge, which dries to form crusts, or the surface becomes studded with large scales. At other times, as at present, the excoriations become covered, or the scales fall off, leaving the condition seen at the present time a most typical picture of the disease. Suitable remedies may remove scales or cause the excoriated patches to skin over, but they will not cure the disease, which extends steadily in a peripheral manner. Sometimes outlying foci are observed. Patches of telangiectasis may also be seen. In order to diagnose the condition from eczema, it is sufficient to note the bright bluish-red colour, the abrupt border which sometimes has a suspicion of induration. As in all skin affections, the mode of evolution and the history of the case must be taken into account.

The patient informed Mr. Clarke that since last seen a fortnight ago considerable "healing" had taken place. This refers merely to the closing of excoriations and the separation of scales. No true healing takes place in the disease, and we must be prepared to find the aspect of the diseased area change from time to time. When it is unobscured, as in the case before us, the appearance is unmistakable.

The only treatment that could be contemplated was complete removal of the breast.

A word should be added. This disease is not limited to the nipple and surrounding skin. The first example of the affection it was his (Mr Clarke's) fortune to see had begun at the margin of the anus, and extended on to the scrotum. The case was shown by Dr. Louis Wickham at the St. Louis Hospital in Paris.

Dr. ABRAHAM confirmed the diagnosis in this case, and added that examination of a scraping, treated with liquor potassæ, under the microscope, would show the bright corpuscles regarded by Wickham and others as psorosperms. He also concurred in the treatment proposed.

Extract from

"THE REMEDIAL VALUE OF CYCLING."

By OSCAR JENNINGS, M.D., Paris,

In the "Medical Annual," 1896.

THE French Academy of Medicine has approved of moderate cycling in mild heart lesions, and there does not seem to be a dissentient voice. Dr. Herschell has called attention to its danger when practised without judgment, and he and others have quoted several instances of sudden death so resulting. In fact, the case is exactly as I anticipated seven years ago; moderate, graduated cycling is free from danger as a pastime to the heart, and may be of the greatest use to it when diseased; but on the other hand, the cycle is no talisman to prevent a man with a weak or diseased heart suffering from his folly, when he happens to make upon it an effort that would have caused a fatal result under any circumstances. The cases mentioned by Dr. Hammond as being suitable for cycling are simple, degenerated conditions of the muscular fibres in dilated hearts, either with or

without compensatory hypertrophy, and in slight valvular affections. Sir J. B. Richardson points out that enfeebled or worn-out arteries are a source of greater danger than feebleness of the heart. Venous enlargement, he adds, seems to be benefited by cycling.

I shall pass over the other states in which cycling benefits by its mechanical action, merely observing as a matter of curiosity, that hernia, strange to say, is benefited by cycling, as I showed by an extensive investigation seven years ago, quoting previous observers who had noted isolated cases, and the answers of several correspondents. One medical man relates that in his own case a hernia entirely disappeared. Another, who was hereditarily disposed to it through father and grandfather, is obliged to wear a truss when taking other exercise, *e.g.*, horse-riding. The appliance being uncomfortable on one occasion, he decided to try and dispense with it, the result being that the bowel remained within the ring, and that he has always ridden a cycle without it since. This point is worthy of note, for the medical papers periodically publish inquiries on the subject, which often elicit replies from the ignorant, that cycling is decidedly improper without a truss, and not safe with it; whereas the truth is exactly the reverse.

THERAPEUTICAL NOTES.

Sulphur Baths, simultaneously with mercurial frictions, in the treatment of syphilis are supported by Grabowski (*Archiv für Dermatologie und Syphilis*), who is upheld by Kaposi and Finger. By this association greater quantities of mercury are said to penetrate the organism, while a salutary influence is exerted upon the nutritive process of exchange. The frictions should be made some hours after the bath.

Acute Gastro-Enteritis of Infants.—Bismuth becomes particularly efficacious when associated with Colombo root. Colombo root, 1 gram., made into an infusion with 75 grams. of water; to this when filtered is added subnitrate of bismuth, 3 gram., and syrup of bitter orange peel, 15 grams. Dose: teaspoonful every two hours.

(*La Presse Méd.*)

THE CLINICAL JOURNAL.

WEDNESDAY, APRIL 22, 1896.

CLINICAL NOTES.

—♦—
WITH DR. POORE
IN THE
**WARDS OF UNIVERSITY COLLEGE
HOSPITAL.**
—♦—

GENTLEMEN,—The first case I show you is one of
Sporadic Cretinism.

The patient is over 17 years of age, and measures only four feet two inches in height. On looking at the photograph taken on admission you will at once notice the stolid look, the thick lips, the stunted limbs, the big abdomen, and recognise them as characteristic of cretinism. As a further evidence of cretinism, we can detect in the patient no indication at all of the thyroid gland. On putting the patient's head back over the pillow I can get the examining fingers below the thyroid and cricoid cartilages, and can feel the rings down to the sternal notch, and I cannot detect with my fingers on either side of the trachea the slightest vestige of a thyroid gland.

This cretin differs in two respects from ordinary cases. There are no fatty tumours above the clavicles which have been described as so common, and the texture of his skin is finer and less wrinkled than in other cretins I have seen. The thickness of the lips and the heavy stolid look noticed in the photograph taken on admission are now almost gone, and in intellectual power he is far ahead of any congenital cretin I have yet seen. You cannot compare this patient in any way to an idiot. He writes as well as most of us, spells correctly, and reads quite difficult words at sight; he can do a very considerable amount of mental arithmetic. I asked him when he came in what three times 3s. 8d. was, and he very promptly told me the correct answer—11s. He was shown yesterday a book in the Hebrew character, and said that he was not a Jew, but knew that it was a

Hebrew book. You have just heard him tell me that he has been reading about the suicide which has occurred in a hospital; he recognises and names pictures of political celebrities, so that he is unlike the ordinary cretin in being not only a politician, but an observant gossip. Another point is that cretins sometimes have peculiar hair. This boy's hair comes out rather unduly, but there is nothing peculiar about it. On his admission to this hospital our diagnosis of cretinism of a sporadic type was made, but not without some natural reservation, and we gave him thyroid extract, beginning with five grains per day, and that resulted in certain changes. The first thing noted was that his pulse and his temperature increased. The output of urea in the urine underwent a tolerably steady increase, and his weight has undergone a tolerably steady decrease. On the 22nd February this patient weighed 76 lbs., and on the 16th March 68 lbs., so that he has lost eight pounds in weight. He has got much more lively, and he is remarkably improved in appearance. There was some suspicion that other members of the family might be in the same condition, but that does not appear to be the case. He has one sister who is very small, and there is a cross of Portuguese and Tamil in their blood, and though they live at Goa they call themselves Portuguese. The Tamils are, I am told, a small race, and when we say that this patient is only four feet two inches in height, the above facts must be taken into consideration. Further, it appears that his father is only five feet in height, and that the mother is a very short woman.

Actinomycosis.

We now come to a case of some interest, one, that is to say, of actinomycosis. The history of the patient is that he was well up to last August, when he began to feel out of sorts, lost his appetite, and got thin. His occupation is that of a nurseryman, and he is engaged in going about the country attending flower shows. Six months ago he came under the care of a doctor in the country with all the symptoms of effusion into the right pleura.

Paracentesis was performed, and the right pleura was found to contain pus, and the next day he coughed up much foul-smelling pus. It was treated in the usual way for what was quite correctly taken to be an empyema on the right side. The opening in this case was very low down indeed, at the extreme base of the lung, so that at one time it was thought to be an hepatic abscess. The opening you see is at the eighth rib, and lies posterior to the mid axillary line. Another point is that the opening has, so to speak, fungated, and is bounded by what would have been called in former days "proud flesh." The important point about the case is this, if you take a lens and watch the pus as it runs away you will see little granular points in the pus, looking like particles of iodoform; and these points, when placed under the microscope, afford us conclusive evidence that we have to deal with a case of actinomycosis, or in English, the ray fungus. Mr. Crookshank, my house physician, has prepared for us under the microscope a beautifully stained specimen showing the fungus. Like many of the other fungi there is a network of mycelium interlacing, which is stained blue; and in this specimen the interlacing is very intricate, the fibres are short and very much twisted. Arising from this mycelial bed are clumps, and these clumps bear upon their surface club-like rays. These clumps have been compared to the head of a daisy, and this arrangement is well shown in a specimen of bovine actinomycosis, which is shown alongside the specimen from our patient. You can come to a very fair understanding of the complaint and a very correct diagnosis without using all these elaborate staining arrangements, which, however, have added very much to our knowledge; but, supposing you find yourself without their aid, you must remember that if you place one of the yellow points shown in this pus between two cover glasses and break it down slightly by pressure, then, without any staining, you can make out the mycelium, and sometimes you can make out the clubs.

The actinomycosis seems to be a great stimulator of inflammation, and the fungus is generally imbedded in a groundwork of leucocytes.

On examining one of these yellow points between two cover glasses, which spread it out, you will see the yellow colour by transmitted light. The diagnosis first rests on the naked eye cha-

racter of these little yellow spots, then on the discovery of the mycelium, and further on staining the finding of the club-like bodies.

It is only of recent years that the occurrence of actinomycosis in the human subject has been proved. It has been known, however, in animals, and is a well-known disease of cows, called by the veterinary surgeons "wooden tongue;" and this wooden tongue was a matter of common knowledge before it was proved to be the result of the ray fungus. In man the ray fungus has been found to affect the lungs, the bones, and the liver. Dr. William Ransom has described a case affecting the rectum and urinary tract, which case got perfectly well under treatment.

With regard to prevention and treatment we must consider, first, how the ray fungus enters the body. Whether it enters by diseased meat I cannot say. If meat be the seat of an actinomycotic growth, and were not properly cooked, it would certainly be a source of danger. There have been some cases which have been attributed to eating barley. The grain and the husk of barley are said to be liable to be affected by the ray fungus. To return to the treatment, many cases of actinomycosis have got better under iodide of potassium. This patient is having large doses of iodide of potassium, 25 grains three times a day. His temperature has been as high as 103° on one occasion, and it has not changed very much since he has been in; but taking it on the whole the later tendency is perhaps a little downwards, and the excursions of the temperature are less pronounced than they were. In addition this man is having one tenth of a grain of silver nitrate three times a day, and I am ordering the granulations at the orifice of the wound to be also rubbed with silver nitrate, and an injection to the wound of 1 grain of silver nitrate to the ounce.

Silver nitrate is inimical to some fungi. What directed attention mostly to this point was the experiments of Raullin, as recorded in Duclaux's book on fermentation, with *aspergillus niger*, a mould which grows on solution containing saccharine matters. Raullin cultivated his *aspergillus* with great care in porcelain trays (like photographic trays), and he found by keeping the temperature at a certain point that he could gather in a crop once in six days. He treated his trays like a farmer works his fields; his estate, however, was

only about six inches square, and he cultivated his aspergillus in a fluid of a certain definite composition. I cannot tell you exactly what it was, but it contained nitrates, salts, sugar, and so forth. The composition of this fluid was changed till a fluid was found that gave the maximum crop of aspergillus. The aspergillus showed a marked aversion to certain substances, and the most marked instance was nitrate of silver. If there was added to the cultivating fluid $\frac{1}{1000000}$ th part of silver nitrate the aspergillus would not grow, neither would it grow in a silver bowl; and beyond any question there is some antipathy to silver on the part of the aspergillus. It is on that account that I have been giving this man silver nitrate internally, and applying it externally. Silver nitrate used to be called and perhaps is so still called the surgeon's friend. Proud flesh in the olden days had silver nitrate well rubbed on it, and no doubt gave the quietus to many an organism working mischief in the tissues. I may also add that the aspergillus was affected much more by silver than by mercury, and by certain other metals with antiseptic properties. With regard to the uses of silver nitrate in medical practice I have found it of very decided use in dilated stomach with fermentation. There was a man last summer in the ward who was supposed to be suffering from cancer of the stomach. His vomit was very foul, and the stomach was dilated; but with nitrate of silver he got what might be styled fairly well, and he left the hospital, and we never could confirm the diagnosis of cancer. It was probably a case of dilated stomach, with unwholesome putrefactive organisms.

Supposed Case of Intestinal Obstruction.

This patient, æt. 19, follows the occupation of a waiter. He came into the hospital yesterday with a history of pains in the abdomen, and on the right side, with vomiting. He has been ill for a week. Two days ago the pain was very severe, and was worse on movement. The patient has had slight pain in the same place before, but has never vomited or been confined to bed. The family history is good. The temperature now is 102°. The man has had beef-tea since coming in, and has not been sick, but he has retched. The stools came away by using an enema, and he has passed wind since, but has not vomited. He has had a belladonna fomentation

over the right side of the abdomen, and his pain is now relieved. No opium has been given, and he now no longer complains of pain.

In many abdominal cases, especially intestinal obstruction, opium has been and is much ordered; but when you give opium in these cases you must remember that the opium effectually masks all the symptoms, and you cannot tell your patient's condition; and if you ask a surgeon to see your patient after you have given him opium he will be unable to form any opinion from the symptoms in the case.

This man you will notice is now lying comfortably in bed. He has been complaining of pain in the abdomen, and has vomited, so we therefore examine the abdomen, and notice that it is not distended. The abdomen is about normal in shape, and pain is complained of in the right iliac fossa and above it. There is no distension or pain anywhere. From the right iliac fossa to the umbilicus the pain yesterday was very marked. On rolling the patient over on his left side some resistance is noted in the right flank, and some hardness is to be felt apparently in the hepatic flexure of the colon, which is in all probability fecal. The temperature has come down, the man has lost his pain, he is feeling comfortable, and his condition is fair. I feel sure that the proper treatment of this case for the next twenty-four hours, or until we get some other symptoms, is to leave the man alone, keep him in bed on spoon diet, beef-tea, milk, and so forth; but give him no drugs at all. I do not order him opium, because he is free from pain, and does not require it. On no account should a man in this condition be given any purgative. I would even go further, and advise that it is undesirable for the present to repeat the enema. Leave the man completely alone, and we shall probably find in twenty-four hours the bowels will recover their tone; and if that is not the case, it will be quite time enough then to consider further treatment. The man is not in an acute state, and the slight traces of albumen in the urine may, I think, for the present be allowed to pass unconsidered.*

Convalescence from Rheumatic Fever.

This is a case of convalescent rheumatic fever

* The patient continued to improve, and left the hospital quite well in a few days.

without any complication, except a little bronchitis. He has been under salicylate of soda, and it must be remembered that as long as patients are under the influence of salicylate you cannot tell how they are really doing. In this case, after 15 grains hourly, the temperature rapidly fell, and since his admission the amount has been gradually decreased. The temperature is now normal, and we assume that he is now convalescent. He has no critical complications, his bronchitis is lessening, and now is the time in an attack of bronchitis to give what often proves a sovereign remedy, namely turpentine. But always be sure when giving turpentine as to the condition of the urine. Here there has been only a trace of albumen once or twice; his work connects him with alcohol, the man being a potman, and possibly there may be some chronic kidney mischief, therefore the effect of the turpentine must in this, as in all cases, be carefully watched. In my opinion there is no remedy like turpentine in the later stages of bronchitis.

Convalescence from Typhoid. Pus in the Urine.

In this bed we have a case of typhoid that is getting well. The temperature since his coming in has gradually fallen, and reached the normal on the forty-fifth day of the illness, as far as we can judge. Since then for ten days the temperature has remained below 99°, and his diet is being slightly improved. First, he had stale bread and butter; now he is having tripe, which is one of the best and most digestible things you can give to a patient who is recovering from typhoid. This man has one complication, it is an accidental complication of his fever; he has been suffering from gonorrhoea with a slight purulent discharge, and it is curious to note that during the fever pus has not ceased to appear in the urine, though it is not at all uncommon for that to occur. When he was sent in and admitted, the pus was thought to be from a secondary infection of the kidney following on the gonorrhoea, and I am not altogether sure that this pus does not come from the kidneys. The amount of pus is considerable, it is certainly more than occurs in an ordinary case of gonorrhoea; and although more pus comes off in the first urine passed than with the last, it is very well diffused throughout the urine. It is possible that he may have an ulceration near the neck of the

bladder, but I am inclined to think that some pus comes from the kidney, because of the acidity and because of the equal diffusion. Of course we have not been able recently to palpate with any force at all, but as far as we can see at present there is no trace of any tenderness in the region of the kidney. On gentle palpation with one hand in front and the other under the loin, I can feel no enlargement nor detect any tenderness on either side. I have asked Mr. Barker to explore the urethra, and it may be that the pus only comes from some local trouble. This patient was passing pus while the fever was upon him, which is not very common. Many samples of urine that I saw were ammoniacal, and I remember one particularly which had not been passed more than an hour, and which was distinctly ammoniacal, and the question arises whether this ammoniacal decomposition has taken place in the bladder or in the vessel after the passing of the urine. It must be distinctly and clearly understood that, if you pass urine into a vessel containing any impurity, decomposition may be very rapid indeed; and if the urine is passed into an impure bed bottle, and if that is then allowed to remain for any time under the hot clothes decomposition is extremely rapid, and I am not prepared to say that the change might not be instantaneous. Here, however, with perfectly clean vessels and the urine passed in the presence of my house physician, we have been able to determine that there is no ammoniacal decomposition. If this urine had been ammoniacal when passed it must have been ammoniacal in the bladder; but although the urine became ammoniacal very soon afterwards, so as to lead to doubt as to whether or not the bladder was at fault, still there was the absence of all symptoms of irritability of the bladder. When the urine is ammoniacal and the decomposition takes place in the bladder, the patient suffers from catarrh of the mucous membrane of the bladder, and there are incessant calls to pass water. In this patient nothing of the sort has happened, but you must also remember that you may have suppuration in the kidney without increased frequency of micturition. In the urine we can find no renal derivatives, except a very occasional cast and one or two blood corpuscles which were noticed in the early part of the case.

WITH DR. HABERSHON
IN THE
OUT-PATIENT DEPARTMENT AT THE
HOSPITAL FOR CONSUMPTION AND
DISEASES OF THE CHEST, BROMPTON.

Pneumothorax.

THIS is a remarkable case, because it is an illustration of an extremely favourable instance of pneumothorax. If you had not seen the man before, and had not known that he had pneumothorax, I do not think that you would detect it. The physical signs are those of fluid in the right side of the chest. You notice the whole right side of the chest is absolutely dull up to the second rib, and above that point it is impaired slightly. The dullness of the right side extends a little beyond the left margin of the sternum, so that the limits of the left pleural cavity are somewhat contracted. The heart's apex beat, however, is not displaced very far to the left. At present it is in the fifth space, a little beyond the nipple line or the middle clavicular line. The physical signs over the dull area on the right side are those of complete absence of breath sounds up to the second rib, and above that point typically weak amphoric breathing. The vocal vibrations are abolished over the lower part of the chest, but increased in the clavicular region above and below. The breathing on the left side of the chest is somewhat harsher than normal, but there are no adventitious sounds there or on the right side. The voice resonance is increased over the whole right side; there is weak, distant pectoriloquy over the lower two thirds of the right side, with a slight ægophonic twang; towards the upper part, immediately beneath and above the clavicle on the right side, there is well-marked pectoriloquy.

The only positive sign of pneumothorax is that the patient has a most marked succussion splash, which varies from day to day on the occasion of his visits, according to the amount of fluid present. This succussion splash is easily heard by putting your ear to the chest and shaking the patient; and this patient tells you that he always hears it himself, and on one or two occasions the splash has been distinctly heard some distance from the

patient. This case is interesting, because the patient has at no time ever presented any marked signs of tubercle. He was taken into the ward with a certain amount of fluid on the right side of the chest and a displacement of his heart to the left, and it was not until he had been in some time that it was discovered that he had pneumothorax, on account of the limited amount of displacement of the heart. His sputum was examined, and tubercle bacilli were found; there was no sharp onset in the symptoms, but it was a gradual development.

This is a wonderful instance of the good results obtained by leaving alone a hydro-pneumothorax. The condition of the patient is very good. He has a good colour, and is not livid; he coughs scarcely at all, and with only occasional expectoration; the only symptom of which he complains is a certain amount of shortness of breath on exertion. In other respects he feels well.

All this shows, then, that the indications for interference with the fluid in a case of pneumothorax are when the dyspnoea is so excessive as to necessitate some operation, or when the general condition of the patient seems to be that produced by absorption of pus. Possibly in this man the fluid is not purulent.

We have had many notable instances of the dangers of removing fluid in such cases. The compression of the tubercular lung seems to have a powerful effect in arresting the tubercular process; when the compression is relieved by taking away the fluid you frequently get an outburst of tubercular inflammation, and not infrequently of a miliary character. One patient now in our wards with pneumothorax lay for a long time under my own observation in another hospital, and finally went away to a home on the sea coast, where the resident medical officer tapped him on several occasions, with the result, I fear, that the patient is now dying from an acute outburst in the remaining lung.

The man I show you to-day is, I think, the best example I have ever seen of hydro-pneumothorax with a favourable prognosis.

Diagnosis between old Gastric Ulcer and
Malignant Disease.

This is a patient, who, when he first came, presented the signs of severe gastric disturbance.

He gave a history of severe epigastric pain after food for the last eight months, and in August of last year he vomited blood. The important part of the case is the question of the diagnosis between old gastric ulcer and malignant disease. The man has lost a considerable amount of flesh, and while he has not got a particularly cachectic appearance, still on the other hand he has in the epigastrium a very definite feeling of resistance immediately below the ensiform cartilage. The diagnosis then rests between the presence of a small malignant tumour of the gastric walls, or a thickening around an old gastric ulcer. The patient has been very much relieved by the medicine containing bismuth ordered for him with a few grains of sulphocarbonate of soda, and it is now clear that the pain is very much better. This, of course, does not determine the diagnosis. I have often seen cases of thickening about a gastric ulcer which have been mistaken for malignant disease, but, on the other hand, we must not forget that malignant disease sometimes follows the occurrence of a gastric ulcer. We had a case here not long ago, in which a chronic gastric ulcer was found with enormously thickened walls, presenting all the appearances of a scirrhus cancer. Against the view that this man is a case of simple gastric ulcer is one fact, that when there is puckering and thickening of a chronic gastric ulcer, it is usually accompanied with dilatation of the stomach, this being a well-known sequela of chronic gastric ulcer. Dilatation more often follows the chronic gastric ulcers, especially in the middle-aged and old, than the ulcers seen in younger people. In this case, the area of stomach dulness is very little increased, and is certainly not enough to give the bell sound on auscultation. Before we can determine whether it is a malignant case we must leave the man somewhat longer, but the material improvement manifested to-day may rather point to the less grave condition.

Mitral Disease.

This is a case of mitral disease, with very considerable hypertrophy of the heart. The apex of the heart is in the fifth space, about an inch beyond the middle clavicular line; the upper limit of cardiac dulness extends to the third costal cartilage, the transverse dulness at this point is distinctly increased to about two inches, and the dulness extends to about a finger's breadth to the

right of the sternum. The impulse is markedly heaving in a patient only 16½ years of age. She has been under my observation for two years since February 7th, 1894. She has an extremely loud and harsh systolic murmur, and an extremely feeble second aortic sound at the apex. This systolic murmur is heard over the left back. On lying down the systolic murmur becomes, if anything, somewhat louder. The second sound is still extremely feeble at the apex, but with it there is now a very short diastolic murmur. The case seems to me to be one of mitral regurgitation, with, in all probability, mitral stenosis. There is no distinct presystolic murmur, and the points in favour of stenosis are that the prolonged murmur begins a little before the time of the systolic cardiac impulse, with an extreme feebleness of the second aortic sound and the diastolic murmur of mitral origin, which is not heard anywhere else except at the apex. This diastolic murmur is usually called the early diastolic bruit of mitral disease, and I always regard it as a suction murmur, but I must leave the question of the mechanics of these murmurs for another day.

This case is an example of a common form of mitral disease, found in young subjects. A large heart with a heaving impulse, and generally marked pulsation over the whole cardiac area, and a double apex murmur, systolic and diastolic. Though there is no distinct presystolic murmur, in all probability we should find a button-hole mitral valve. Hypertrophy is always a marked sign in young subjects, because though (as Dr. Gowers has pointed out), time is always a necessary factor in the production of hypertrophy, a much shorter time is needful in youth on account of the greater activity both of growth and nutrition, so that repair and an attempt at compensation is more easily produced.

The chief point in treatment is to deal with the precordial pain that she suffers from. I believe this pain is due to intercostal neuralgia, from the pressure of the hypertrophied heart on the ribs. It is to be noted that the pain she complains of is always in the same place. I have previously tried her with plasters, and again she is now trying a belladonna plaster.

Diagnosis of the size of the Right Ventricle in a case of Mitral Stenosis.

This forms a very good parallel case with the

other heart case we have just had. It is also one of my old cases. There is a presystolic thrill to be felt. The heart is extremely enlarged, and the apex is in the anterior axillary line in the sixth space. The cardiac dulness extends upwards to the lower margin of the second rib. In the second interspace the transverse dulness is about two inches in length, from about a quarter of an inch to the right of the sternal border to an inch beyond the left sternal border. The right margin of the cardiac dulness is an oblique line extending from the lower part of the second costal cartilage down to the liver dulness, to a point about three-quarters of an inch from the right border of the sternum. There is great heaving of the whole cardiac area, and in this case also, there is a very prolonged murmur at the apex, but the presystolic element is very rough. The presystolic part blends with the systolic portion, and the second sound in this case is almost inaudible. The second pulmonary sound is very much increased.

There are one or two points of great interest in this case. There seems to be very little doubt that part of the cause of the feebleness of the second aortic sound is due to the pushing backwards of the left ventricle by the increasing dilatation of the right ventricle. Of course, a large part of the cause for the weakness of the second aortic sound is the low tension in the left ventricle, and feeble closure of the aortic cusps; but there is another important point in this case, and that is, what means have we of telling what is the size of the right ventricle, and how far it has pushed back the left ventricle? I believe, and always teach that in these cases we can diagnose by the character of the second sound the extent of the enlargement. At the apex, which is in the anterior axillary line, you will hear the murmurs blending, and you will hear a very feeble second sound that continues till you get to about the normal nipple line, but beyond that point you at once hear a loud clapping second sound. I believe you have crossed the septum, and you are no longer listening over the left ventricle, but you are listening to the sounds from the right ventricle. The presystolic and the systolic murmurs are heard, but more muffled, and the second sound is heard with great clearness. Compare the second sounds at the base, and you will hear a weak aortic and an accentuated second pulmonary sound. We have all the physical signs

to indicate dilatation as well as hypertrophy of the right ventricle. There is a soft systolic murmur heard to the right of the sternum, there is a little flickering in the jugular vein, and the patient has some lividity of the ears especially, which are deeply red, and there is blueness of the lips, all signs of increased pulmonary engorgement. On making the patient lie down, the murmur to the right of the sternum becomes more typical, the flickering in the veins more marked, and leaves no doubt that the murmur is due to tricuspid regurgitation. It is not possible to say whether the pulse is single or double in the veins. There is also epigastric pulsation.

The case is one that is often described as the third, or late stage of mitral stenosis, in which the right ventricle is not sufficiently hypertrophied to overcome the block at the mitral valve, and has dilated. The features of this case are of an intermittent dilatation of the right ventricle, succeeded by a recovery under treatment. There is evidently mitral regurgitation and stenosis, with a moderate secondary tricuspid regurgitation.

I shall order this man the ammoniated gentian mixture of the hospital, adding to it *Mvij* of digitalis, for seven days, and direct that the patient should be kept in bed.

Cardiac Pain.

There are many cases of mitral stenosis in which cardiac pain is the chief symptom complained of, and it is one of the commonest complaints heard from patients in the out-patient department.

The commonest position for pain is under the left clavicle, though in these cases to-day the pain was complained of in the cardiac area. You will note that both were young cases with considerable hypertrophy of the heart, and I have little doubt but that the great size of the heart causes compression on the intercostal spaces, giving rise to an intercostal neuralgia. I have another case under my care of great hypertrophy of the heart, associated also with pain and extreme tenderness in the region below the left breast; and in this case too, I believe, the cause of the pain to be the great pressure of the hypertrophied heart.

Amyloid Disease, with Contraction and Retraction of the Left Lung.

This is a case of amyloid disease. There is an

old contracted cavity in the left side of the chest, and the heart is drawn up to the second rib. There is undoubted amyloid trouble, with a considerable amount of albuminuria, with diarrhoea at times.

We were talking about retraction of the lungs and displacement of the heart, and we saw one case a short time back of rapid contraction in a year and a half, with the heart drawn over from the left side to the right, but not displaced upwards. This particular case we are examining now has an excavation in the left apex, the heart is drawn up almost to the second rib, the right margin of the cardiac dulness commencing about an inch and a half from the left border of the sternum, and extends right across the chest a little below the line of the second rib, and then blends with the dulness over the left half of the chest and in the left axillary portion; but the heart can be felt beating as far as the anterior axillary line, and you can feel its heaving character. The tumultuous beating of the heart prevents any very definite apex beat being made out. The signs of the cavity were previously well marked, but are now obscured. The breathing is weak over the greater part of the lung, but there is still some weak amphoric breathing at the left apex. Above the left clavicle there are well-marked signs of a cavity, loud cavernous breathing, the rales are very large and gurgling, and there is post-tussic suction. The left side of the chest is extremely dull.

It is a very good case of great contraction and retraction of the left lung. The other physical signs point to amyloid disease. On deep breathing you can feel the spleen in this patient.

Transposition of Viscera.

There is no special interest about this patient, except as a rather unusual anomaly, and in point of physical signs an excellent trap for the unwary. It is a case of transposition of the viscera. She has some chronic bronchial catarrh, and is in rather feeble health and of a somewhat neurotic temperament. The heart is almost entirely on the right side of the chest, the liver dulness is found exactly transposed, the stomach is on the right side instead of the left, and the spleen also is on the right side.

Hæmoptysis or Hæmatemesis?

Female patient, æt. 30. There is a consumptive history in this case. She says that she has coughed up blood, and the last occasion on which it occurred was seven days ago.

This patient is a good illustration of one of those cases in which it is extremely difficult to determine whether the blood is from the lung or from the stomach; and it is generally found that, where vomiting also is a symptom in the account, the difficulty of being sure, from the statements of the patient, of the origin of the blood is very greatly increased. The mere act of vomiting may cause the patient to cough; and if there be hæmatemesis, some of the blood may find its way into the trachea, and from thence it may be coughed back. On the other hand, if a patient has considerable hæmoptysis, some of the blood may be swallowed, and then be vomited. To determine this point it is necessary to consider the other signs and symptoms in a case, looking mainly to those symptoms which would indicate whether the lungs or the stomach are affected. Our patient here tells us that she feels sick after food, and that she has severe pain for twenty minutes after eating. She has then pain after food, no vomiting, but nausea and retching in the morning; but against these indications must be placed her cough and expectoration, with a strong family history of phthisis.

One must not forget, also, that gastric ulcer is not by any means uncommon in cases of phthisis. I collected 200 cases some years ago from my father's notes of gastric ulcer, and in this series of cases 2 per cent. only had phthisis. Other authorities, however, give the percentage of cases of gastric ulcer which have also tubercular disease of the lung as somewhat higher. In spite of all this, it is extremely improbable that she could have had hæmoptysis continuing at frequent intervals during the last year or nine months, without presenting marked symptoms of lung disease. But as far as I can discover her chest is quite healthy, perhaps there may be a little harsh breathing at the right apex, but generally it is vesicular. There are no adventitious sounds anywhere even after coughing. On examining the epigastrium you noticed that the patient flinched, and it is likely from the tenderness that there has been some ulceration of the stomach in this case. With a bad family history of phthisis it would be very unwise to send this patient into the hospital even though she is so unwell, because we ought not to put her in a consumptive atmosphere.

Her diet should be a liquid one, milk and a little soda water, broths of any kind—mutton broth, or veal broth, or rabbit broth—but not beef tea. Thicken the milk with a little arrow-root, if it can be taken without causing pain.

I shall order her our bismuth mixture with an addition of morphia:

R	Bismuthi subnitritis	gr. xx
	Misturæ tragacantha	f 3ss
	Tincturæ cinnamomi	℥ x
	Liq. morphinæ hydrochloratis	℥ v
	Aq.	ad 3j

INDEX TO VOLUME VII.

	PAGE		PAGE		PAGE
A		Apnoea and dyspnoea, case of, with aneurysm	113	Bougies, how to pass	23
Abdominal muscles, case of congenital absence of	220	Apoplexy, lecture on a case of	135	Boxall, Dr. R., on general management of labour, etc.	165, 185
Abdominal section, after-treatment of	251	Appendicitis, operation or not? statistics of course of	320	Bradford, Dr. J. R., on disseminated sclerosis	323
Abductor paralysis not always fatal	229	Arsenic and herpes	311	Brain, features of ventricular hæmorrhage in	136
Abortion, threatened and missed	117	" " lupus	366	Braine, Mr. C. C., fatal case of hæmorrhage under anæsthesia	359
Abscess, caecal, and subjective smell	55	" " curious case of poisoning by eruption from	357	Breast, case of Paget's disease of	391
" ischio-rectal	69	Arthritis, traumatic, lecture on	245	Breasts, the, care of in suckling	190
" pelvic, from hip disease	210	" " tubercular endo- v. peri-articular	210	" " in new-born babies	190
Acromegaly, anatomy of	226	" " formation of abscess in	210	Bright, Dr., and the kidney	54
" differential diagnosis of	226	" " frequency of	209	Brodie, Mr. C. G., on congenital dislocation of the hip	84
" lecture on	225	" " lecture on	209	Bromoform in whooping cough	224
" pathology of	226	" " multiple	211	Bronchitis and turpentine	396
" symptoms of	225	" " site of	209	" herpetic, treatment of	192
" treatment of	227	" " treatment of	212	" in typhoid	50
Acromion, frequency of fracture of	250	Ascites, lecture on	289	" subacute, formula for	208
Acro-scleroderma following Raynaud's disease	240	" treatment of	291	Bryant, Mr. T., clinical cases	112
Actinomycosis, case of	393	Aseptic, thioform as an	350	Bullet-wound of skull, case of	92
" fungus of	394	Aspergillus and silver nitrate	395	Burse, the, infection of, in hip-disease	210
" treatment of	394	Asthma and sneezing	368	Butlin, Mr. T. H., on treatment of cancer by injection	213
Addison's disease, case of suspected	389	Atheroma, nature of	114	Buxton, Dr. D., on pental	342
Adenoids, tracheotomy in a case of	229	Atropine and thyroid secretion	97		
Adenoma of endometrium	377	" duration of action of	42		
Æther or chloroform?	344	Auto-intoxication	48		
Albuminoid disease, symptoms of	146				
Albuminuria and Bright's disease	53				
" " eclampsia	11				
" " mercury	53				
" " pental	343				
" diet in	13				
Albumoses	383, 384				
Alcohol and delirium, lecture on	58				
" " insanity, lecture on	329				
" " phthisis	304				
" " urethral stricture	21				
" " uterine hæmorrhage	119				
Allchin, Dr. W. H., lecture on the nature of disease	70				
Alumol in gonorrhoea	44, 382				
Amyloid disease, case of, with lung trouble	399				
" " without albuminuria	11				
Anæmia and plumbism	193, 195				
" formula for gastric troubles	219				
Anæsthesia in thoracic aneurysm	299				
" fatal case of hæmorrhage under	359				
Anæsthetics in tracheotomy	230				
Analgin in malaria	28				
Aneurysm, case of, with dyspnoea	112				
" thoracic, bruits in	298				
" " causes of	301				
" " lecture on two cases of	297				
" " physical signs of	297				
" " reflex signs of	300				
" " symptoms of	297				
" " treatment of	301				
Aniline dyes and cancer	218				
Ankle-clonus in disseminated sclerosis	324				
Antilactagogue, cocain as	344				
Anus, fissure of	67				
" treatment of pruritus of	68				
Aphonia and chronic laryngeal catarrh	294				

G	PAGE
Galabin, Dr. A. L., lecture on hysterectomy	102
Gall-bladder <i>v.</i> right renal tumour	101
Gall-stones, case of, causing intestinal obstruction	92
Gangrene, pathology of	115
" senile, case of	114
Gastro-enteritis, formula for	392
Giddiness as a symptom	129
Glands, case of inflamed cervical	37
Glands, case of symmetrical enlargement of	239
" inguinal, case of tubercle of	37
" invasion of, by sarcoma of testis	150
Glaucoma, contraindication of mydriatics in	43
Glioma of cord <i>v.</i> brain	14
Glycerophosphates, indications for	60
Goitre, exophthalmic, cardiac bruits in	39
" " lecture on	38
" " pathology of	39
" " treatment of	40
Gonorrhœa, alumol in	44, 382
Gout and lysisid	344
" " plumbism	91, 195
Gowers, Dr. W. R., caries of spine	17, 33
" " poliomyelitis, adult	313
" " " infantile	241
G. P. I. and grandiose delusions	357
" knee-jerks in	355
" lecture on early	355
" " <i>v.</i> cerebral syphilis	356
Grant, Mr. D., on diagnosis of pain in and about the ear	5
Graves' disease, lecture on a case of	93
" " symptoms of	98
" " treatment of	94, 95
" " unilateral features of a case	96
Guaiacol in typhoid	32
Gums, blue line on, due to charcoal	375
Gunn, Mr. R. M., on external examination of the eye	137

H	PAGE
Hadley, Dr. W. J., lecture on a case of acromegaly	225
Hæmatemesis, treatment of	164
" ulcer of stomach <i>v.</i> cirrhosis of liver	161
" " <i>v.</i> hæmoptysis	400
Hæmaturia absent in renal sarcoma	100
Hæmophilia and uterine hæmorrhage	119
Hæmoptysis and phthisis	305
" " <i>v.</i> hæmatemesis	400
Hæmorrhage, fatal case of, under anæsthesia	359
" " in endometritis	378
" " intravenous injection in	360
" " uterine, lecture on	117
Hæmostatic, dermatol as	224
Hair dyes and lead	200
" the, in cretinism	393
Hall, Dr. A. J., lecture on exophthalmic goitre	38
Hall, Dr. F. de H., lecture on gastric ulcer	161
Hands, lotion for chapped	14
Harley, Dr. Geo., on structure and formation of calcoli	30
Hay fever, formula for	208
Heart, case of fibroid patch in	146
" " disease and blue pill	56
" " case of	223, 398

Heart disease, case of, advanced	PAGE
" " diagnosis of size of right ventricle in	387
" " lecture on	27, 145
" " pain in	398, 399
" " some typical cases of	27
" " possible influence of syphilis on	145, 147
Heath, Mr. C., common diseases of rectum	65
" " stricture of rectum	202
Hemianæsthesia in disseminated sclerosis	325
Hemianopsia in neurasthenia	284
" " temporal	225
Hemiplegia, relation to paralysis agitans	293
Hepatitis, malarial, treatment of	44
Herman, Dr. G. E., on endometritis	377
Hernia after abdominal section	252
" " anomalies of testis in relation to	286
" " Bassini's operation for	221
Herpes and arsenic	311
" " case of relapsing, from syphilis	255
" " zoster, notes on a case of	311
Hip bursæ, about, and tubercle	210
" " disease, first symptoms of	345
" " " how to measure in	347
" " lecture on	345
" " ulceration in	348
" " effect of traumatism on	249
" " dislocation of congenital, lecture on	84, 90
" " " features of	85
" " " operations for	88
" " " pathology of	86
Hoarseness and cancer of larynx	267
Hutchinson, Mr. J., clinical cases	28, 254, 321, 357
Hydrastis in uterine hæmorrhage	122
Hydrostomia, case of	29
Hyoscin and insomnia	387
Hyperidrosis in Graves' disease	93
Hyperpyrexia, case of	388
Hypnotic, chloralose as	60
Hysterectomy, details of	104
" " lecture on	102
" " varieties of operation for	103

I	PAGE
Ichthyosis, case of congenital	224
Incubation period and ferments	384
Infant, newborn, management of	189
Influenza as excitator of mania	333
" " case of	386
Insanity after general anæsthesia	330
" " alcoholic, forms of	329
" " lecture on	329
" " symptoms of	333
" " prognosis in	338
" " of muscles	15
Insomnia and hyoscin	387
" " trional in	44
Intermittent fever and tablets of quinine	208
Intestine, obstruction of, case of	92
" " in peritonitis	349
" " supposed case of	395
Intestines, fæcal obstruction of	391
Intravenous injection in hæmorrhage	360
Iodine in gastric catarrh	360
Iodoform and pulse rate	254
Iris, description of	139
Iritis, mydriatics in	41
Irrigation in abdominal operations	83

J	PAGE
James, Dr. A., lecture on pontine lesions	125
Jamison, Dr. W. A., lecture on psoriasis	154
Jaundice, causes of obstructive	124
" " remarks on theory of	122
" " hæmatogenous <i>v.</i> hepatogenous	123
Jennings, Oscar, on cycling	392
Joints affected by posture and pressure	245, 246
" " lecture on tubercle of	209

K	PAGE
Kaposi's disease, two cases of	254
Keratosis, senile, of hands and feet, case of	321
Kidney, affections of, as prognostic in myelitis	111
" " case of fixation of	235
" " inflammation of	390
" " sarcoma of	98
" " of absence of symptoms in	100
" " of microscopic appearances of	101
" " of physical signs of	100
Kidneys granular at 18	223
" " in plumbism	195
Knee, case of loose body in	375
Knee-jerk and sensory nerves	313
" " in G.P.I.	337, 355
" " in neurasthenia	284
" " in spinal injury	282

L	PAGE
Labour, anæsthetics in	170
" lecture on general management of	165, 185
" " melancholia after	76
" " support of perineum in	170
" " vaginal examination in	169
Lachrymal gland, how to examine	138
Lane, Mr. W. A., on traumatic arthritis	205
Lardaceous disease, case of	399
" " symptoms of	146
Laryngitis, chronic, treatment of	369
" " case of	368
Laryngotomy, considerations of	230
Larynx, cancer of, age in	267
" " always primary	265
" " appearances of	269
" " diseases resembling	274
" " divisions of	266
" " duration of	274
" " etiology of	265
" " extrinsic, origin of	273
" " immobility of cords in	271
" " lecture on	265
" " may be pedunculated	270
" " microscopic appearances of	274
" " operations for	276
" " pain in	268
" " progress of	272
" " prognosis of	276
" " sex in	266
" " situation of	271
" " smoking in	266
" " symptoms of	267
" " <i>v.</i> innocent tumours	270
" " <i>v.</i> papilloma	271
" " <i>v.</i> syphilis	275
" " <i>v.</i> tubercle	274
" " case of syphilis of	387

	PAGE
Phthisis , formula for dyspnoea in . . .	264
" insidious origin of . . .	303
" lecture on beginning of . . .	302
" sudden onset of . . .	305
Physiology , definition of . . .	73
Piles , symptoms of . . .	65
" treatment of . . .	65
" " sphincter in . . .	66
Pilocarpine and thyroid secretion . . .	97
" in diabetes . . .	344
" not to be used in . . .	
eclampsia . . .	13
Placenta , management of . . .	187
Pleura , great effusion in, in cancer of . . .	
lung . . .	89
" effusion in, and phthisis . . .	397
Pleurisy and phthisis . . .	306
Plica , semilunaris . . .	138
Plumbism , an obscure case of . . .	197
" and gout . . .	195
" and monoplegia, case of . . .	90
" changes in organs in . . .	195
" diagnosis of . . .	196
" from cider drinking . . .	199
" lecture on . . .	193
" symmetry of lesions in . . .	198
" symptoms of . . .	193
" treatment of . . .	200
Pneumonia and phthisis . . .	306
" digitalis in . . .	208
Pneumothorax , case of . . .	397
" succussion splash in . . .	397
Poisoning by arsenic, curious case of . . .	57
Poliomyelitis , adult, lecture on . . .	241, 313
" " pain in . . .	242
" treatment of . . .	316
Polypi , aural, causes of . . .	339
" " lecture on . . .	339
" " symptoms of . . .	339
" " treatment of . . .	339
" " varieties of . . .	339
Pons , lecture on lesions of . . .	125
" symptoms of lesions of . . .	125, 127
Ponsonby's drops . . .	51
Poore, Dr. G. V. , lecture on chronic . . .	
lead poisoning . . .	193
Powder for new-born babies . . .	189
Pregnancy , hæmorrhage in connection . . .	
with . . .	117
" operations during . . .	65
Presystolic bruit , case of . . .	295
Processus vaginalis testis . . .	286
Proctotomy for stricture of rectum . . .	205
Prurigo and coal tar . . .	344
" hyemalis . . .	254
Pruritus ani , treatment of . . .	68
" scrotal, treatment of . . .	60
Psoriasis , age in . . .	154
" anatomy of . . .	156
" and scarring . . .	156
" " tinea versicolor . . .	156
" diagnosis of . . .	156
" frequency of . . .	154
" lecture on . . .	154
" margin of, when spreading . . .	155
" of nails, case of . . .	255
" preputial . . .	133
" pathology of . . .	156
" rupoides, case of . . .	28, 155
" situation of . . .	154
" spread of . . .	154
" treatment of . . .	157
" <i>v. syphilis</i> . . .	156
" varieties of . . .	155
Ptyalism , case of chronic . . .	29
Puerperium , when to close . . .	19
Pulmonary artery , rupture of aneu- . . .	
rysm into . . .	301
Pulse , the, in aneurysm . . .	114, 300
" " and iodoform . . .	254
" " " temperature after ab- . . .	
dominal section . . .	253

	PAGE
Pulse, the, in peritonitis	350
Pupil, the, in hæmorrhage	360
" guide to administration of opium	319
Pupils, consensual activity of	140
Purpura hæmorrhagica following eczema	321
Pus in urine after typhoid	396
" consideration of	396
Pyæmia and ear disease	8
Pyelo-nephritis as prognostic in myelitis	III
Pyoktanin and cancer	341

Q

Quain, Sir R., reminiscences	51
Quinine for cough	192

R

Railway-spine	283
Raynaud's disease with severe hæ-	
morrhage	322
" " followed by acro-	
scleroderma	240
Reaction of degeneration, the . .	194
Rectum, the, blood from, in children .	69
" " in neurasthenia	285
" " lecture on common	
diseases of	65
" " lecture on stricture of . .	202
" " means of dilating stric-	
ture of	204
" " morning diarrhoea in	
cancer of	205
" " operations for stricture	
of	206
" " prolapse of	69
" " stricture of, from epithe-	
lioma	205
" " stricture of, from syphilis	
tallow candle as dilator	
of	204
Recurrent laryngeal nerve and	
aneurysm	299
Reflexes in cord disease	111
" " v. compression	21
Refraction, use of mydriatics in esti-	
mation of	42
Reminiscences by Sir R. Quain . .	51
Review, Ashby and Wright (children)	
Carter (medicine)	32
Dermatological Society	176
Gowers (clin. lectures)	124
Hausen and Loogt (leprosy) . . .	31
Harris and Beale (phthisis) . . .	192
Jones (electricity)	256
Kelly's Directory	328
Lang (eye examination)	224
Lewis (scoliosis)	256
Lindsay (malaria)	224
Luff (forensic medicine)	31
Marsh (joints)	296
Morris (genito-urinary)	256
Neale's Digest	208
Phonographic Record	160, 312
" outlines	311
Stewart, Physiology	31
Sajous' Annual	159
Stocken (dental)	16
Sturges (heart)	296
Temperance Hospital Reports . .	32
Year Book of Treatment	256
Rheumatism and chorea	15
" " sciatica	24

	PAGE
Rheumatism, case of acute	386
" convalescence from,	395
" case of	257
Rheumatoid arthritis and injury	246
" " micro-organisms of	360
Rhinorrhœa and sneezing	238
Ringworm, followed by general der- matitis, case of	27
Roberts, Dr. F. T., lecture on heart disease	98
Robson, Mr. A. W. M., on sarcoma of kidney	239
Rodent ulcer, peculiar form of	122
Rolleston, Dr. H. D., on jaundice	117
Routh, Dr. A., on uterine hæmorrhage	155
Rupia psoriasis	355
Russell, Dr. J. R., on G.P.I.	355

S

Sacralgia	242
Sarcoma of omentum, case of	390
Sarcomata, case of multiple	28, 255
" of kidney, treatment of	98
" of testis, forms of	152
" "withering"	28
Scalp, dry seborrhœa of	192
Sclerosis, disseminated, age in	323
" " anatomy of	324
" " apoplexy in	326
" " causes of	323
" " death in	328
" " diagnosis of	327
" " eye muscles	327
" " in	325
" " fits in	326
" " hemianæsthesia	325
" " lecture on	323
" " optic neuritis	327
" " in	326
" " sex in	323
" " speech in	325
" " symptoms of	324
" " traumatism in	323
" " types of	324
" " v. cerebral tumour	328
Sclerotic, characters of	139
Scopolamine as mydriatic	431
Scrofuloderma, case of	240
Sebaceous tumours becoming malignant	357
Seborrhœa, treatment of	192
Semon, Dr. F., on malignant disease of larynx	265
Serum and antitoxins	384
" treatment of variola	191
Shoulder, effect of traumatism on	250
Silver, nitrate of, and aspergillus	395
Skull, case of bullet-wound of	92
" fracture of	35
Small-pox, serum treatment of	191
Smell, case of morbid subjective	55
Smith, Dr. R. S., morbus cordis, with hepatic enlargement	145
Sneezing, paroxysmal, case of	367
" treatment of	368
Snell, Dr. S., use of mydriatics	41
Snow, Dr., on cholera	51
Spasms and pental	343
Spasticity, cause of	110
Specialism and exclusiveness	17
Specifics, origin of	216
Spencer, Dr. H. R., on ruptured perineum	307
Spermatic cord, enlargement of in growths of testicle	151
Sphincter ani, stretching of, in piles	66

W		PAGE			PAGE			PAGE
Waldo, Dr. H., on chorea . . .	15		Warts, formula for . . .	224		White, Dr. W. Hale, thoracic aneurysm	297	
" " on myelitis . . .	109		" v. epithelioma . . .	131		" " on peritonitis . . .	317	
" " on ascites . . .	289		Water and cholera . . .	51		Whooping cough, bromoform in . . .	224	
Walsh, Surg.-Capt., on dysentery . . .	261		" " lead . . .	200		Wilkin, Mr. G. C., on aurial polypi . . .	339	
Warner, Dr. F., on ulcerative endocarditis . . .	257		Watson - Williams, Dr. P., Graves' disease . . .	93		Winter cough and phthisis . . .	304	
			Weir Mitchell treatment, what is it ? . . .	285		Worms and dysentery . . .	262	
			West, Dr. S., on alcoholic delirium . . .	58		Wrist-drop . . .	194	
						Writer's cramp simulated by torticollis	219	

END OF VOLUME VII.

The Clinical Journal, April 22, 1896.]

PRINTED BY EDWARD KNIGHT, MIDDLE STREET, E.C.



1 gal
5-17

